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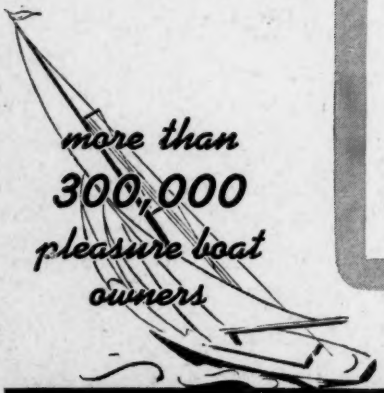

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 25 JULY, 1942 NUMBER 7

THE TREATMENT OF SYPHILITIC PRIMARY OPTIC ATROPHY*

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INTRODUCTION

In 1932, one of us (J. E. M.) brought up to date the literature on syphilitic primary optic atrophy.[1] Recently (1940), Moore and Woods[2, 3] presented critical reviews of the pathology and pathogenesis of this condition, and of the results of treatment. In 1938, Moore and others[4] published a preliminary report concerning the results of treatment of our own clinical material.

MATERIAL

The present study consists of an evaluation of the results of treatment in 250 cases of syphilitic primary optic atrophy seen in the Syphilis Clinic of the Johns Hopkins Hospital and in the private practices of two of us (J. E. M. and A. C. W.) from 1918 to 1940, inclusive. A few additional verified cases were available for analysis as to subsidiary points, although absence of adequate anamnestic data or follow-up examinations prevented their utilization for the evaluation of treatment.

These patients were drawn from a much larger material of some 500 cases, the remainder of which were discarded because of diagnostic study too incomplete to insure differentiation between primary and secondary optic atrophy, or, if certainly primary, to insure that syphilis and not some other condition was the cause; or because of complicating ophthalmologic features, such as extensive chorioretinitis, cataract, glaucoma, or possible tryparsamide or alcohol amblyopia. The ophthalmologic diagnosis in all cases was established by a member of the Department of Ophthalmology of the Johns Hopkins Hospital. In the statistical analysis that follows, the tables and discussion include data on all those patients with verified primary optic atrophy with regard to whom information on the point considered was available. The length of this analysis precludes the presentation of individual case reports, although the actual clinical observations on three of the patients included in this study are presented in "The modern treatment of syphilis."[5]

* From the Syphilis Division of the Medical Clinics, and the Wilmer Ophthalmological Institute, the Johns Hopkins University and Hospital. This paper is published simultaneously in the American Journal of Syphilis, Gonorrhea, and Venereal Diseases and in the American Journal of Ophthalmology.

† This study, begun some 15 years ago by Drs. Moore and Woods in collaboration, has been participated in by many members of the staffs of the Syphilis Division and the Wilmer Ophthalmological Institute, to whom grateful thanks are due. In the present report, the responsibility for clinical data as to syphilis is assumed by Dr. Moore, for ophthalmologic details by Dr. Woods, for careful visual field studies by Dr. Sloan, and for the excellent statistical study and preparation of the manuscript by Dr. Hahn. A joint responsibility is, of course, shared by us all.

TABLE 1

DISTRIBUTION OF CASES OF OPTIC ATROPHY BY RACE, SEX, AND DIAGNOSIS

Race	Sex	Total	Tabes	Tabo-paresis	General Paresis	Diffuse Meningo-vascular	Con-genital
White	Male	129	65	16	13	27	8
	Female	18	7	0	0	10	1
Colored	Male	94	38	10	3	41	2
	Female	23	6	1	2	12	2
Total		264	116	27	18	90	13

The association of primary optic atrophy with various types of neurosyphilis, and its distribution by race and sex. It is a matter of common neurologic and ophthalmologic knowledge that primary optic atrophy is more frequently associated with tabes dorsalis than with other forms of neurosyphilis; and that in this condition the optic atrophy (and the associated pupillary changes) is often the first evidence of central-nervous-system damage to appear. "Preataxic" optic atrophy is such a common phenomenon as to require no documentation. In our own material, however, the diagnosis of tabes dorsalis has not been made in any case unless, associated with optic atrophy and pupillary changes, there were also present definite neurologic evidences of damage to the posterior columns of the spinal cord or unless such manifestations developed after the appearance of optic atrophy. Even with these diagnostic restrictions it is shown in tables 1 and 2 that in our material, as in that of others, optic atrophy occurs more than twice as frequently in tabes as in other types of neurosyphilis.*

This association of optic atrophy with the spinal-cord changes of tabes dorsalis is especially striking when the incidence of optic atrophy in general paresis and taboparesis is considered. Of the 885 patients classified in table 2 under paresis and taboparesis, 757 were paretics without obvious evidence of spinal-cord involvement; of these only 18 (2.4 percent) had primary optic atrophy. Of the 128 taboparetics, no less than 27 (21.1 percent) had optic atrophy. This association of optic atrophy with taboparesis is so striking as to suggest that its occurrence in paretics without spinal-cord involvement must be exceedingly rare. In our own material, the existence of tabes combined with paresis in the 2.4 percent of cases showing apparently pure paresis with optic atrophy may well have been missed by incomplete neurologic examination in which the finer evidences of cord involvement were overlooked.

In the recent neurologic and ophthalmologic literature alike, there are many references to primary optic atrophy associated not with tabes, but instead with

* The figures given in table 2 represent the minimum rather than the actual incidence of optic atrophy in various types of neurosyphilis. If all dubious and complicated cases had been included (almost equal in number to those actually shown), an equally false maximum picture would have been shown. The actual incidence probably is half again as high as shown; that is, about 20 percent of all patients with tabes and about 12 percent of all patients with acquired symptomatic neurosyphilis may be expected to develop optic atrophy, complicated or otherwise.

so-called "syphilitic basilar meningitis." The 90 cases included in table 2 among "other forms" of neurosyphilis (excluding parenchymatous, asymptomatic, and congenital) would be so classified by many clinicians. In these cases, the diagnosis of neurosyphilis hinges on the existence of optic atrophy and pupillary changes in patients who, as a rule, showed no other demonstrable neurologic damage either to brain or spinal cord that would justify the diagnosis of tabes or paresis. In our own Clinic, such patients have been classified as "diffuse meningovascular neurosyphilis," in the absence of clinical evidence permitting more accurate pathologico-anatomic localization. This classification is adhered to throughout this paper in order to determine by study of the various points involved whether such a neurologic separation from tabetic primary optic atrophy is justified. Interestingly enough, in view of the frequent mention in the literature of "pre-ataxic optic atrophy," no patient in this group has been observed to develop subsequent evidence of tabes dorsalis, although a number, on the basis of a final rather than the original neurologic diagnosis, have ultimately developed paresis (and have therefore been classified in tables 1 and 2, among the paretic group).

It has repeatedly been stated in the literature that primary optic atrophy due to "basilar meningitis" is more amenable to treatment and, in general, offers a better prognosis than the tabetic form. More recently it has been suggested that in paresis the lesion in the optic nerve is predominantly infiltrative and inflammatory in contrast to the degenerative lesion observed in tabes; and that in paresis also, the prognosis of optic atrophy should be better than in tabes. These assumptions are wholly unjustified on the

TABLE 2

INCIDENCE OF OPTIC ATROPHY IN DIFFERENT TYPES OF NEUROSYPHILIS BY RACE AND SEX

		Total	White Male	White Female	Colored Male	Colored Female
Tabes	Number of cases	849	469	108	199	73
	Number with optic atrophy	116	65	7	38	6
	Percentage with optic atrophy	13.7	13.9	6.5	19.1	8.2
Paresis and Taboparesis	Number of cases	885	509	162	159	55
	Number with optic atrophy	45	29	0	13	3
	Percentage with optic atrophy	5.1	5.7	0	8.2	5.5
Other Forms excluding Asymptomatic and Congenital	Number of cases	1459	436	231	495	297
	Number with optic atrophy	90	27	10	41	12
	Percentage with optic atrophy	6.2	6.2	4.3	8.3	4.0
Total	Cases of neurosyphilis*	3193	1414	501	853	425
	Number with optic atrophy	251†	121	17	92	21
	Percentage with optic atrophy	7.9	8.6	3.4	10.8	4.9

* Symptomatic, exclusive of congenital.
† Not including 13 patients in whom optic atrophy was associated with congenital neurosyphilis.

basis of pathologic studies of the optic pathways in the patients with the several types of neurosyphilis exhibiting clinical evidence of optic atrophy. There are comparatively few satisfactory pathologic observations of primary optic atrophy in any type of neurosyphilis, and such as are available are wholly inadequate to permit of conclusions as to types of optic atrophy associated with different types of neurosyphilis. Whether there is clinical evidence justifying a different prognosis of optic atrophy in patients with paresis, tabes, or apparently diffuse meningovascular neurosyphilis will be considered later.

At the moment, however, we may anticipate by saying that we believe there is no justification for the diagnosis of basilar meningitis in neurosyphilitic patients with optic atrophy as something apart from tabes or paresis, or both. In both these conditions there is usually present some degree of basilar meningitis; and the existence of this particular lesion is accepted by some (Ingvar[6]) as the cause of the pupillary abnormalities associated with tabes, paresis, and other forms of neurosyphilis. Since the optic nerves also course superficially and in close contact with the meninges, it is believed by many that primary optic atrophy is due to the meningeal lesion in all forms of neurosyphilis. It is true that this explanation is not entirely adequate either for optic atrophy or pupillary changes since, in many neurosyphilitic patients with optic atrophy, no evidence of existing or previous basilar meningitis can be found at necropsy; and, in the majority of those in whom it is present, optic atrophy does not occur.

Whatever the cause of optic atrophy may be, however, and whether due to the mild basilar meningeal involvement almost universal in all forms of neurosyphilis, to a hypothetically postulated toxin, or to a combination of syphilis and nutritional deficiency,[2] it seems to us probable that the fundamental process is the same in all types of neurosyphilis, and that no distinction is to be made between optic atrophy in tabes and paresis and in diffuse meningovascular neurosyphilis in which the tabetic and paretic syndromes are (perhaps only temporarily) absent.

The breakdown of our material by diagnosis, race, and sex discloses an important, and so far as we know, hitherto undescribed fact (table 2). The distribution of our cases of neurosyphilis shows, as in previous reports from our own Hospital and those of many other observers, a preponderance of white patients over Negroes (which, considered in the light of the total syphilitic population at our disposal, amounts to nearly 3 to 1), and a preponderance of males of both races over females in the proportion (population groups considered) of 5 to 1. Primary optic atrophy is, however, more frequent among neurosyphilitic Negroes of both sexes than among neurosyphilitic white patients, the percentage incidence for Negro males being 10.8 as contrasted to 8.6 for white males, and the incidence for Negro females being 4.9 as contrasted to 3.4 for white females. The proportionately higher incidence is shown for all types of neurosyphilis and the sample of persons is not only random but sufficiently large to be convincing. This racial selectivity of optic atrophy is especially unusual since both tabes and paresis themselves are much more common in whites.

Is optic atrophy on the increase or decrease? It has been suggested that as a result of the more adequate treatment of syphilis in recent years, optic atrophy is

on the decrease. From our own Clinic, as table 3 shows, no such decrease is apparent. During five 4-year periods from 1921 to 1940, inclusive, the number of cases of neurosyphilis has not changed significantly as a whole (nor in any of its various diagnostic types). Likewise, there appears to have been no significant decrease in the relative incidence of optic atrophy. Indeed, the proportion of cases abruptly doubles after 1925. This probably does not represent an actual increase, however, because our interest in the problem of optic atrophy as a cooperative undertaking between the Departments of Ophthalmology and Syphilis became a major one in about 1925. Many more patients were referred to the ophthalmologist for special study, and consequently more diagnoses of early optic atrophy were made.

The spinal-fluid findings as an aid to the classification of the neurologic type of optic atrophy. With a view to examining the validity of the diagnosis of optic atrophy due to "basilar meningitis" as opposed to that due to tabes, aid has been sought from the spinal-fluid findings. These data are presented in table 4. The classification of cerebrospinal-fluid changes is that of Moore, as follows: Group I, minimal alterations—that is, pleocytosis with or without protein increase but with cerebrospinal-fluid Wassermann and colloidal tests negative; group II, intermediate changes with some degree of Wassermann fixation in the fluid; group III, maximal changes—the "paretic formula." As might be expected,

TABLE 3

INCIDENCE OF NEUROSYPHILIS AND OF OPTIC ATROPHY BY 4-YEAR PERIODS—1921 TO 1940, INCLUSIVE

Period	Total Cases of Neurosyphilis, Excluding Asymptomatic	Total Cases of Optic Atrophy	Percentage with Optic Atrophy
1921–1924	628	34	5.4
1925–1928	570	69	12.1
1929–1932	685	58	8.5
1933–1936	522	56	10.7
1937–1940	621	44	7.1

all but 2 of the 37 patients with paresis or taboparesis show the paretic formula in the cerebrospinal fluid, and in all the fluid is definitely positive. Interest lies in the comparison of optic atrophy in tabes and in diffuse meningovascular neurosyphilis. Here the proportion of fluid changes of varying types is approximately the same in the two conditions. Obviously, therefore, tests of the cerebrospinal fluid fail to differentiate the hypothetical two types of optic atrophy. It is worth remarking that the cerebrospinal fluid was completely normal in nearly 7

TABLE 4

COMPARISON OF CEREBROSPINAL FLUID IN PATIENTS WITH OPTIC ATROPHY IN TABES, DIFFUSE MENINGOVASCULAR NEUROSYPHILIS, PARESIS, AND TABOPARESIS

Diagnosis	Number of Cases	Percentage with Negative Spinal Fluid	Percentage with Group I Spinal Fluid	Percentage with Group II Spinal Gluid	Percentage with Group III Spinal Fluid
Tabes	97	10.3	2.1	27.8	59.8
Diffuse Meningovascular	87	5.8	2.3	28.7	63.2
Paresis and Taboparesis	37	0	0	5.4	94.6
Total	221	6.8	1.8	24.4	67.0

TABLE 5

RELATIONSHIP OF DURATION OF INFECTION* TO APPEARANCE TIME OF OPTIC ATROPHY

Duration of Infection to Onset of Optic Atrophy (years)	Less than 5	6–10	11–15	16–20	21–26	27–30	31+	Duration Unknown (History Negative)
Number of patients with parenchymatous neurosyphilis	3	13	21	22	10	4	5	70
Number of patients with diffuse meningovascular neurosyphilis	1	9	11	14	5	0	4	46
Total patients	4	22	32	36	15	4	9	116

* Based on presumptive history of early syphilis.

percent of all cases, the diagnosis being, nevertheless, clear on the basis of a positive blood serologic test and of unmistakable neurologic evidence of neurosyphilis other than optic atrophy.

The relationship of a history of early syphilis to the development of optic atrophy. Almost 50 percent of the 238 patients concerning whom data were available had no recollection of any of the manifestations of early syphilis. Of 36 females, only 3 (8 percent) had a presumptive history of early syphilis, while of 202 males, such a history was obtained in 119 (59 percent). Of the 148 patients in whom optic atrophy was associated with parenchymatous neurosyphilis, 78 (53 percent) had a history of early syphilis; and of the 90 patients with so-called meningovascular neurosyphilis, a similar history was obtained in 44 (49 percent).

In table 5 is shown the duration of infection for the 122 patients with respect to whom it was possible to estimate this factor. There is a wide scatter, ranging from 3 to 44 years after infection. These data also fail to differentiate the supposed two types of optic atrophy, for in both the parenchymatous and meningovascular group approximately 55 percent of all cases appeared between the eleventh and twentieth year after infection, and the average duration of infection in each group was approximately 14 years.

The identical appearance time of optic atrophy and the similar findings in the cerebrospinal fluid lend support to the opinion previously expressed that this process is fundamentally identical in the two groups of cases. Moreover, its close clinical association with tabes suggests that the lesion is fundamentally parenchymatous. There remains for subsequent consideration the question of prognosis.

The relationship of treatment for early syphilis to the development of optic atrophy. Of 235 patients in whom this factor could be estimated (table 6), only

TABLE 6

RELATIONSHIP OF OPTIC ATROPHY TO TREATMENT FOR EARLY SYPHILIS

Type of Treatment for Early Syphilis	Number of Patients with Optic Atrophy Receiving Specified Treatment	Percentage of Patients Receiving Specified Treatment
None	188	80.0
Oral, or heavy metal only	15	6.4
Less than six doses arsphenamine	21	8.9
7 to 20 doses arsphenamine	8	3.4
More than 20 doses arsphenamine	3	1.3
Total	235	100.0

3 (1.3 percent) had received anything approaching adequate treatment for early syphilis; only 32 (13.6 percent) had received any arsphenamine product at all; and 188 (80 percent) had received no treatment of any kind. These data suggest strongly that, as for other lesions of late syphilis, the adequate treatment of early syphilis confers nearly complete protection against the development of optic atrophy.

Methods of treatment

In general, our material is subdivided into six groups as follows:

(1) Eighty-nine patients who received no treatment at any time.

(2) Thirty-seven patients who received only inadequate routine treatment arbitrarily defined as less than 10 injections of a trivalent arsenical and 10 injections of a heavy metal or the equivalent thereof.

(3) Forty-two patients who received adequate routine treatment; that is, more than 10 injections each of an arsenical and a heavy metal.

(4) Thirty-two patients whose treatment was initiated with one or more subdural intraspinal or intracisternal injections of arsphenaminized serum by the Swift-Ellis technique, this form of treatment usually being either accompanied or followed by routine treatment.

(5) Eleven patients whose treatment was initiated by induced malaria followed by routine treatment.

(6) Thirty-nine patients treated by a combination of two or more of these systems. Sixteen of these patients were given routine treatment followed by induced malaria, in turn followed by subdural therapy in four instances. Fifteen patients were given routine treatment followed by subdural therapy, in turn followed by induced malaria in three instances. In two patients, treatment was initiated by induced malaria, which was followed by subdural therapy; and in the remaining six patients treatment was initiated by subdural therapy, which was followed by induced malaria. It was obviously not always possible to evaluate all types of treatment for each individual patient. The method and rationale of approach to the evaluation of this group of patients forms an integral part of the evaluation of the various types of treatment, and will become clear subsequently as each group is considered separately.

The basic data concerning each of the aforementioned groups of patients are presented in tables 7, 8, 9, 10, and 11. Table 7 shows, for the untreated group, the neurologic diagnosis, the interval from onset of symptoms to date of observation, the visual acuity in both eyes at the time of the last observation, and the type and degree of visual-field defect when known. Tables 8, 9, and 10 show, for the routine, subdural- and malaria-treated groups, respectively, the neurologic diagnosis, duration of symptoms, and visual acuity at the start of treatment when known, kind and amount of treatment, duration of observation after treatment, total duration of observation, visual acuity at the end of the observation period, and the progress of visual-field changes when known. Table 11 shows similar data for patients treated by a combination of methods. For this group the duration of symptoms and visual acuity after each type of treatment is recorded.

Criterion of blindness

For purposes of comparison, blindness in both our untreated and treated patients is defined as central visual acuity of 10/200 or less in the better eye. In both groups visual acuity at the time of observation is whenever possible expressed

TABLE 7

DATA ON 89 PATIENTS WITH OPTIC ATROPHY WHO RECEIVED NO TREATMENT AT ANY TIME

History Number	Diagnosis	Interval from Onset of Symptoms to Recorded Observation (months)	Visual Acuity		Type and Degree of Visual-Field Change when Known
			Better eye	Worse eye	
1) W.B.	Tabes	1	L.P.	L.P.	
2) K 14563	Paresis	2	0	0	
3) K 77732	Diffuse meningovascular	2	5/200	L.P.	
4) J 4542	Tabes	2	16/200	0	
5) 205728	Tabes	3	20/70	20/100	I Moderate
6) F 81676	Diffuse meningovascular	3	20/40	20/200	
7) 120580	Taboparesis	3	1/200	1/200	
8) H 70726	Paresis	3	H.M.	C.F.	
9) J 66472	Diffuse meningovascular	4	L.P.	L.P.	
10) U 172578	Tabes	4	0	0	
11) K 52154	Diffuse meningovascular	4	L.P.	0	
12) K 97641	Diffuse meningovascular	4	0	0	
13) K 78470	Diffuse meningovascular	4	H.M.	0	
14) 114826	Tabes	4	0	0	
15) G 35217	Tabes	4	C.F.	0	
16) 131120	Tabes	4	4/200	0	II Advanced
17) 137276	Congenital	5	0	0	
18) H 88534	Diffuse meningovascular	5	10/200	3/200	
19) H 67543	Paresis	5	0	0	
20) 203125	Tabes	6	C.F.	L.P.	
21) U 10893	Diffuse meningovascular	6	0	0	
22) H 83452	Tabes	6	5/100	1/200	
23) H 61474	Paresis	6	20/40	L.P.	
24) K 32018	Diffuse meningovascular	6	L.P.	L.P.	
25) 166057	Taboparesis	6	5/100	5/200	I Advanced
26) K 16341	Tabes	7	20/100	20/200	II Advanced
27) U 26201	Diffuse meningovascular	8	0	0	
28) J 52579	Tabes	9	H.M.	0	
29) 108054	Congenital	9	0	0	
30) U 42676	Tabes	9	20/200	20/200	IV Moderate
31) 102888	Paresis	10	C.F.	0	
32) H 34605	Diffuse meningovascular	10	H.M.	H.M.	
33) G 0664	Paresis	10	20/50	0	
34) U 40712	Diffuse meningovascular	11	10/200	2/200	IV Advanced
35) H 33843	Tabes	12	0	0	
36) K 80479	Tabes	12	C.F.	0	
37) J 1034	Diffuse meningovascular	12	0	0	
38) K 45338	Congenital	12	0	0	
39) G 59022	Diffuse meningovascular	12	20/100	0	
40) K 73351	Diffuse meningovascular	12	C.F.	0	
41) 113405	Congenital	12	20/30	20/100	IV Moderate
42) K 14319	Tabes	12	L.P.	L.P.	
43) U 36308	Taboparesis	12	5/200	0	
44) H 89956	Tabes	12	5/200	H.M.	
45) H 64337	Tabes	12	6/200	L.P.	
46) 19097	Tabes	12	L.P.	L.P.	
47) 101585	Diffuse meningovascular	12	8/200	6/200	IV Advanced
48) H 45946	Paresis	12	20/100	20/100	
49) U 52021	Diffuse meningovascular	12	0	0	
50) 114865	Taboparesis	12	H.M.	0	
51) 108508	Diffuse meningovascular	12	4/200	L.P.	IV Advanced
52) J 12038	Diffuse meningovascular	14	0	0	
53) J 6945	Tabes	14	20/15	20/30	
54) J 28029	Tabes	15	L.P.	L.P.	
55) G 53916	Diffuse meningovascular	15	0	0	
56) K 91582	Diffuse meningovascular	18	C.F.	0	
57) G 78048	Tabes	18	20/20	N.D.	
58) 153238	Diffuse meningovascular	18	L.P.	0	
59) K 890	Congenital	18	L.P.	0	
60) H 64343	Diffuse meningovascular	18	2/100	L.P.	

TABLE 7 (*Continued*)

History Number	Diagnosis	Interval from Onset of Symptoms to Recorded Observation (months)	Visual Acuity		Type and Degree of Visual-Field Change when Known
			Better eye	Worse eye	
61) H 27558	Diffuse meningovascular	19	20/70	H.M.	II Moderate
62) G 61498	Tabes	19	0	0	
63) 48705	Tabes	20	0	0	
64) J 15770	Tabes	22	H.M.	H.M.	
65) 187681	Diffuse meningovascular	24	C.F.	0	
66) J 37495	Tabes	24	0	0	
67) J 88542	Taboparesis	24	20/40	20/50	I Slight
68) G 42088	Tabes	24	L.P.	L.P.	
69) 159446	Diffuse meningovascular	24	20/100	H.M.	I Moderate
70) G 99993	Diffuse meningovascular	24	L.P.	L.P.	
71) H 84940	Tabes	24	C.F.	C.F.	
72) H 31397	Tabes	24	C.F.	C.F.	
73) 133692	Tabes	30	0	0	
74) K 30396	Diffuse meningovascular	36	0	0	
75) J 48553	Tabes	36	L.P.	L.P.	
76) G 97967	Diffuse meningovascular	36	3/200	L.P.	
77) J 31107	Congenital	36	15/70	0	
78) K 20041	Paresis	36	4/200	L.P.	
79) U 48609	Tabes	36	C.F.	0	
80) 185894	Tabes	37	20/100	2/200	I Moderate
81) R.B.	Paresis	48	0	0	
82) 132980	Diffuse meningovascular	60	4/200	L.P.	II Slight
83) 213572	Diffuse meningovascular	54	20/30	20/50	I Advanced
84) D.B.	Tabes	54	20/200	4/200	II Advanced
85) 194414	Diffuse meningovascular	96	0	0	
86) J 23431	Diffuse meningovascular	72	0	0	
87) K 97699	Diffuse meningovascular	144	C.F.	0	
88) G 43221	Tabes	180	20/200	L.P.	
89) 220884	Diffuse meningovascular	210	20/200	C.F.	I Advanced

Abbreviations: L.P. = light perception only; H.M. = hand movements only; C.F. = counts fingers at five feet.

The explanation of visual-field classification is found in the text under the section on visual fields (see table 20).

as corrected rather than uncorrected. We recognize the shortcomings of utilizing only central visual acuity as a measure of useful vision, which is better estimated by a consideration of both central visual acuity and visual fields.[7, 8]

The inclusion of the type and degree of visual-field defect in our statistical analyses has not, however, proved feasible for several reasons. First, it is difficult to express such change numerically in a form suitable for such comparative analysis. Second, the data available for many patients do not permit an accurate evaluation of visual-field changes. Obviously, no statement can be made as to type of visual-field change in patients already blind at the time of our first observation, and there are many of these in our untreated group. Unfortunately, also, in many treated patients we do not have accurate fields charted by the same observer before and after treatment. A study of individual case records has revealed such variation in both peripheral and central fields, as charted by different observers, as to preclude their inclusion in a statistical analysis based upon objective data. It has therefore seemed wise to express progress of the optic atrophy in the simplest possible terms—central visual acuity as measured by the Snellen chart. Since the criterion of blindness is the same in both treated and

TABLE 8

DATA ON 79 PATIENTS WITH OPTIC ATROPHY TREATED WITH ROUTINE ANTISYPHILITIC TREATMENT ONLY

History Number	Diagnosis	Interval from Onset of Symptoms to Start of Treatment (months)	Visual Acuity before Treatment		Treatment	Summary of Treatment: Adequate or Inadequate	Observation Period After Treatment (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Change Before and After Treatment When Known		Remarks
			Better eye	Worse eye					Better eye	Worse eye	Before treatment	After treatment	
1) I.B.	Tabes	ND	ND	ND	30 Bismarsen elsewhere	Adequate	ND	36	20/100	1/100	ND	II Advanced	
2) J 70111	Tabes	6	ND	ND	13 mos. elsewhere	Adequate	18	24	H.M.	0			
3) 177821	DMV	8	20/20	20/100	20 Arsphenamine; 38 Bismuth	Adequate	66	74	20/30	3/200	II Moderate	Unchanged	
4) S.M.	GP	ND	ND	ND	Routine 2 yrs. elsewhere	Adequate	ND.	17	H.M.	L.P.	ND	II Advanced	
5) 214579	Tabes	6	ND	ND	30 Arsphenamine; 30 Bi.	Adequate	11	17	20/200	20/200	ND	II Moderate	
6) H 60504	Tabes	ND	ND	ND	22 Arsphenamine elsewhere	Adequate	ND	15	0	0			Treatment begun when V. O.D. was fair
7) J 43637	Tabes	3	ND	ND	Routine 1 yr. elsewhere	Adequate	12	15	4/200	3/200			Treatment said to have begun soon after onset of symptoms
8) 219649	DMV	24	20/200	20/200	8 mos. elsewhere	Adequate	8	32	L.P.	L.P.			
9) U 5639	Tabes	0	ND	ND	22 As.; 40 Hg elsewhere	Adequate	14	14	3/200	0			Treatment at onset
10) K 5388	Tabes	No symptoms	20/15	20/15	19 As.; 9 Bi.	Adequate	41	41	20/15	20/15	II Moderate	No change	
11) K 35939	GP	2	ND	ND	16 As. plus additional treatment	Adequate	28	30	0	0			
12) R.K.M.	DMV	ND	ND	ND	9 mos. elsewhere	Adequate	ND	8	L.P.	0			Treatment at onset
13) U 20854	GP	15	ND	ND	17 As.	Adequate	9	24	L.P.	L.P.			
14) H 53783	Tabes	ND	ND	ND	16 As. elswhere	Adequate	ND	5	20/200	L.P.			
15) L.H.W.	Tabes	17	ND	ND	15 Maph.; 50 Bi.	Adequate	19	36	20/20	L.P.	ND	I Advanced	Had 2 artificial fever treatments
16) C.M.F.	Tabes	2	20/20	20/20	20 As.	Adequate	3	5	2/50	2/70	III Moderate	III Advanced	
17) G 83814	Tp	12	20/50	20/50	6 mos. plus 1 I.S.	Adequate	6	18	20/40	20/50			
18) U 64266	Tabes	ND	ND	ND	50 Bi.	Adequate	ND	11	1/200	L.P.			
19) K 60028	DMV	1	20/20	ND	20 mos. (of this 16 mos. elsewhere; 13 As. here)	Adequate	25	26	20/30	L.P.			

TABLE 8—*Continued*

History Number	Diagnosis	Interval from Onset of Symptoms to Start of Treatment (months)	Visual Acuity before Treatment		Treatment	Summary of Treatment: Adequate or Inadequate	Observation Period After Treatment (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Change Before and After Treatment When Known		Remarks
			Better eye	Worse eye					Better eye	Worse eye	Before treatment	After treatment	
20) U 55148	Tabes	ND	ND	ND	Routine 1 yr.	Adequate	12	12	5/200	L.P.	ND	IV Advanced	
21) G 80334	DMV	7	20/20	20/20	Routine 3 yrs.	Adequate	36	43	20/15	20/20	I Moderate	I Moderate	
22) U 62754	DMV	12	20/200	5/200	Routine 2½ yrs.	Adequate	114	126	8/200	C.F.	ND	IV Advanced	
23) J 8824	DMV	12	20/40	20/70	Routine 1 yr.	Adequate	30	42	20/50	20/70	IV Moderate	Slightly worse	
24) 136500	DMV	12	20/20	20/40	Routine 2 yrs.	Adequate	188	200	20/30	20/30	ND	I Slight	
25) U 58548	GP	17	20/200	C.F.	Routine, 27 mos.	Adequate	22	39	8/200	0	IV Advanced	Worse	
26) K 54477	DMV	4	ND	ND	16 As.; 7 Bi. elsewhere	Adequate	10	14	0	0			
27) H 95972	Tabes	6	15/100	15/100	Routine 1½ yrs.	Adequate	18	24	16/200	16/200			Rapid failure slowed by routine treatment
28) K 53238	DMV	22	2/200	C.F.	40 As.; 32 Bi.	Adequate	54	76	4/200	L.P.	IV Advanced	IV Advanced	Blind before treatment
29) G 48634	DMV	30	10/200	4/200	Routine irregular 2 yrs.	Adequate	22	52	5/200	2/200	I Moderate	ND	
30) H 55467	Tabes	36	20/20	20/120	16 Bi.; 34 As.; 1 I.S.	Adequate	64	100	4/200	2/200	II Moderate	Worse	
31) 159304	DMV	No symptoms	20/15	20/40	12 As.; 8 Bi.	Adequate	8	8	20/15	20/30	I Moderate	I Slight	
32) 115208	DMV	3	20/100	20/200	Routine 2 yrs.	Adequate	38	41	"Useful"	"Useful"	III Advanced	ND	Reads and gets about alone
33) H 79601	Tabes	40	20/50	0	52 As.; 40 Bi.; 1 I.S.	Adequate	44	84	20/100	0			
34) 131123	Tabes	2 (?)	ND	ND	Routine 6–12 mos.	Adequate	ND	Varies 2–6 yrs.	2/200	0			
35) K 35690	Tabes	3	ND	ND	6 mos. As. and Bi.	Adequate	12	15	0	0			
36) 178121	DMV	5	ND	ND	13 As.; 5 Bi.	Adequate	8	13	H.M.	L.P.			
37) 199654	Cong.	ND	ND	ND	19 As.	Adequate	31	31	20/30	20/50	ND	I Slight	
38) 200857	Tabes	48	ND	ND	6 Bi.; 14 As.	Adequate	24	72	2/200	L.P.			
39) U 3084	Tp	1	ND	ND	24 As.; 5 Bi.	Adequate	13	14	L.P.	L.P.			
40) J 90361	Tabes	36	20/20	0	2 yrs.	Adequate	108	144	20/20	0	II Slight	II Moderate	

TABLE 8—*Continued*

History Number	Diagnosis	Interval from Onset of Symptoms to Start of Treatment (months)	Visual Acuity before Treatment		Treatment	Summary of Treatment: Adequate or Inadequate	Observation Period After Treatment (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Change Before and After Treatment When Known		Remarks
			Better eye	Worse eye					Better eye	Worse eye	Before treatment	After treatment	
41) W.L.S.	Tabes	ND	ND	ND	4 yrs. I.M. elsewhere	Adequate	ND	48	4/200	0	ND	IV Advanced	
42) J 46533	DMV	ND	ND	ND	20 As.; 8 Hg elsewhere	Adequate	ND	36	0	0			Treatment said to have begun at onset
43) J 80160	Tabes	See Remarks			8 As.; 12 Bi.	Inadeq.	5	5	H.M.	H.M.			Treatment said to have begun at onset of symptoms
44) J 40433	Tabes	ND	ND	ND	"Few needles" elsewhere	Inadeq.	ND	6	0	0			
45) K 36335	Tabes	ND	ND	ND	3 mos. I.M. elsewhere	Inadeq.	ND	7	L.P.	0			
46) J 48662	DMV	ND	ND	ND	16 Bi. elsewhere	Inadeq.	ND	9	10/200	0			Treatment said to have begun at onset
47) J 17849	GP	2	ND	ND	3 Bi. elsewhere	Inadeq.	8	10	0	0			
48) K 73800	DMV	8	20/70	ND	6 Bi. elsewhere	Inadeq.	38	46	L.P.	0			
49) 111706	DMV	1	ND	ND	9 As. elsewhere	Inadeq.	9	10	0	0			
50) H 65913	Tabes	1	ND	ND	3 As.	Inadeq.	2	3	20/120	C.F.			
51) U 7256	Tabes	7	ND	ND	2 As. elsewhere	Inadeq.	4	11	15/200	4/200			
52) J.S.	Tp	ND	ND	ND	Few Bi.	Inadeq.	ND	24	20/30	20/30	ND	I Advanced	
53) K 38522	Tabes	ND	ND	ND	"Few needles" elsewhere	Inadeq.	ND	12	0	0			
54) K 57807	Cong.	6	ND	ND	2 As. elsewhere	Inadeq.	6	12	0	0			
55) J 98977	DMV	ND	ND	ND	9 As. elsewhere	Inadeq.	ND	6	H.M.	H.M.			
56) H 64867	Tabes	2	ND	ND	6 As.; 2 Bi. elsewhere	Inadeq.	22	24	5/200	0			
57) K 75210	Cong.	ND	ND	ND	6 As.; 8 Bi. elsewhere	Inadeq.	ND	14	0	0			
58) 191930	DMV	4	20/20	6/200	9 Maph.	Inadeq.	8	12	20/20	C.F.	II Slight	I Moderate	
59) H 59670	Tabes	ND	ND	ND	6 As. elsewhere	Inadeq.	12	17	20/200	L.P.			
60) J 52004	Tabes	4	20/30	20/50	6 As.; 13 Bi.	Inadeq.	111	115	20/20	20/50	I Slight	I Advanced	
61) K 25961	GP	ND	ND	ND	8 As.; 8 Bi.	Inadeq.	ND	5	6/200	3/200	ND	II Advanced	

TABLE—8 *Continued*

History Number	Diagnosis	Interval from Onset of Symptoms to Start of Treatment (months)	Visual Acuity before Treatment		Treatment	Summary of Treatment: Adequate or Inadequate	Observation Period After Treatment (months)	Total Observation Period (months)	Acuity		Type and Degree of Visual-Field Change Before and After Treatment When Known		Remarks
			Better eye	Worse eye					Better eye	Worse eye	Before treatment	After treatment	
62) G 50520	DMV	ND	ND	ND	"11 needles" elsewhere	Inadeq.	ND	24	3/100	0	ND	II Advanced	
63) H 51669	Tabes	ND	ND	ND	8 As. elsewhere	Inadeq.	ND	24	L.P.	L.P.			
64) 126464	DMV	ND	ND	ND	10 As.	Inadeq.	ND	8	L.P.	L.P.			
65) J 62925	DMV	ND	ND	ND	"Needles 2 mos." elsewhere	Inadeq.	ND	18	L.P.	0			
66) G 64501	Tabes	ND	ND	ND	Spinal treatment elsewhere	Inadeq.	ND	24	L.P.	L.P.	ND	I Advanced	No data as to "spinal treatment." Statement could not be verified
67) U 39033	DMV	12	ND	ND	10 Bi. elsewhere	Inadeq.	12	24	20/40	20/200			
68) K 24774	Tabes	1	20/70	L.P.	4 As.	Inadeq.	1	2	L.P.	L.P.	I Advanced	0	
69) K 93976	DMV	ND	ND	ND	6 As. elsewhere	Inadeq.	ND	5	0	0			
70) J 52791	Tabes	No symptoms	20/15	20/15	15 Bi.; 2 As.	Inadeq.	24	24	(20/15)	(20/15)	I Early	ND	Final visual acuity estimated. No visual symptoms
71) W.R.P.	Tabes	1 week	20/20	20/20	6 Neo.	Inadeq.	1	1½	0	0			
72) W.B.	Tabes	2	ND	ND	6 As. elsewhere	Inadeq.	3	5	3/100	3/200			
73) E.L.T.	DMV	1	ND	ND	8 As.	Inadeq.	3	4	20/30	20/30	ND	II Moderate	
74) K 93734	Tabes	1	20/20	20/20	4 Neo.; 25 Bi. elsewhere	Inadeq.	17	18	20/100	L.P.	ND	IV Mode rate	
75) J 34385	DMV	4	20/40	L.P.	Tryparsamide 4 mos.	Inadeq.	78	82	L.P.	L.P.			
76) H 52364	DMV	6	ND	ND	3 As.	Inadeq.	6	12	0	0			
77) 158662	Tabes	2	16/100	16/200	8 As.; 6 Bi.	Inadeq.	4	6	0	0	I Advanced	0	
78) 120533	Tabes	ND	ND	ND	8 As.; 10 Bi.	Inadeq.	ND	30	7/200	H.M.			
79) 107802	Tabes	No symptoms	ND	ND	8 As.; 8 Bi.	Inadeq.	27	27	20/50	0			Onset during treatment

Abbreviations: DMV = Diffuse meningovascular; GP = General paresis; Tp = Taboparesis; Cong. = Congenital; ND = Not determined; As. = Arsenical; I.S. = Intraspinal; H.M. = Hand movements only; L.P. = Light perception only; C.F. = Counts fingers at five feet.

TABLE 9

DATA ON 32 PATIENTS WITH OPTIC ATROPHY TREATED INITIALLY WITH SUBDURAL ARSPHENAMINIZED SERUM, WITH OR WITHOUT ROUTINE TREATMENT

History Number	Diagnosis	Duration Optic Atrophy to Start of Subdural Treatment (months)	Number of Subdural Treatments	Visual Acuity before Treatment		Observation After Subdural Treatment (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Changes Before and After Treatment When Known		Remarks
				Better eye	Worse eye			Better eye	Worse eye	Before treatment	After treatment	
1) M.C.A.	DMV	No symptoms	10 I.S.	20/15	20/15	216	216	20/15	20/15	Normal	Normal	Plus tryparsamide and 10 yrs. routine treatment
2) U 2335	Tabes	No symptoms	6 I.S.	20/30	20/70	120	120	20/50	20/50			Plus routine treatment. Final visual acuity based on letter from patient: "Ability to read newsprint"
3) U 42492	DMV	No symptoms	4 I.C.	20/20	Complicated	102	102	10/200	—	IV Moderate	IV Advanced	Plus 2½ yrs. routine treatment
4) U 66530	DMV	2	8 I.S.	ND	ND	16	18	10/200	3/200			Plus 6 mos. routine. Treatment started elsewhere. Visual acuity at start not known
5) U 53781	DMV	3	1 I.S.	20/200	H.M.	1	4	5/200	H.M.	I Advanced	ND	Plus 4 As.
6) U 36812	Tabes	3	13 I.C.	20/50	20/70	117	120	20/70	L.P.	II Moderate	II Moderate	Plus 15 mos. routine
7) R.C.	GP	4	14 I.S.	20/70	2/200	60	64	0	0	IV Moderate	0	
8) W.R.A.	Tabes	4	8 I.S.	20/20	20/50	252	256	20/20	Complicated	I Moderate	I Moderate	All treatment given elsewhere, but records here complete
9) U 40745	Tabes	5	2 I.S.	20/70	20/200	1	6	0	0			Blindness precipitated by intraspinal treatment
10) U 54761	DMV	5	1 I.S.	10/200	L.P.	62	67	12/200	L.P.	II Advanced	No change	
11) G 43589	Tabes	6	4 I.S.	ND	ND	18	24	L.P.	L.P.			Plus 19 As. elsewhere. ND as to visual acuity at start
12) U 37352	Tabes	7	2 I.C.	20/30	L.P.	40	47	0	0	I Advanced	ND	Outside report. Exact date blindness unknown
13) I.K.	GP	8	12 I.S.	20/20	H.M.	180	188	20/20	0	I Advanced (right) Normal (left)	0 (right) I Slight (left)	Plus routine treatment
14) U 23139	Tabes	8	5 I.S. Blind with 3 I.S.	20/30	3/100	4	12	L.P.	L.P.			Visual failure precipitated by intraspinal treatment

TABLE 9—*Continued*

History Number	Diagnosis	Duration Optic Atrophy to Start of Subdural Treatment (months)	Number of Subdural Treatments	Visual Acuity before Treatment		Observation After Subdural Treatment (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Changes Before and After Treatment When Known		Remarks
				Better eye	Worse eye			Better eye	Worse eye	Before treatment	After treatment	
15) U 5410	G.P.	No symptoms	4 I.S. elsewhere	ND	ND	4	4	0	0			Onset visual failure 2 wks. after 1st intraspinal treatment
16) U 48750	Tabes	36	7 I.S.	ND	ND	29	65	20/20	20/20	ND	I Moderate	Plus 35 Diarsenol
17) U 7904	GP	12	1 I.S.	20/60	20/100	24	36	0	0			Based on history and complete atrophy at autopsy
18) U 50031	DMV	19	6 I.S.	20/30	0	92	111	20/30	0	I Moderate	I Moderate	Plus 16 As.
19) U 45924	Tabes	8	8 I.S.	20/30	20/200	129	137	20/15	20/100			Plus 30 As.
20) 138908	DMV	9	8 I.S.	20/100	0	10	19	L.P.	L.P.	II Advanced	L.P.	Plus 1 yr. routine
21) U 6656	GP	9	7 I.S.	20/200	5/100	47	56	5/200	L.P.	II Moderate	II Advanced	Plus 2½ yrs. routine
22) U 36135	Tabes	12	9 I.C.	20/40	0	120	132	10/40	0	IV Early	ND	Plus 1½ yrs routine
23) M.M.	Tabes	15	3 I.S.	20/40	10/100	78	93	20/50	10/100			Plus routine treatment
24) 181936	Tabes	24	8 I.S.	20/20	0	6	30	20/20	0			Plus 20 As.; 5 Bi.
25) U 36974	Tabes	36	4 I.C.	15/200	Complicated	55	91	10/100		III Moderate	III Moderate	
26) 217176	Tabes	36	6 I.C.	20/60	0	177	213	20/70	0	I Moderate	I Moderate	Plus 2 mos. routine
27) U 39055	Tabes	36	2 I.C.	20/200	L.P.	78	114	20/200 ?	L.P.	I Advanced	ND	Final report based on patient's statement
28) U 26163	Tabes	36	5 I.S.	20/200	4/200	22	58	0	0			Plus 2¾ yrs. routine treatment
29) H 32999	Tabes	48	4 I.S.	ND	ND	42	90	L.P.	L.P.			Visual acuity at start of treatment unknown
30) H.	GP	64	5 I.S.	20/20	0	1	65	0	0	I Advanced	0	Blindness precipitated by intraspinal treatment
31) U 65890	Tabes	No symptoms	4 I.S.	20/40	20/40	7	7	20/40	20/40	II Moderate	II Moderate	
32) U 42649	GP	18	3 I.C.	20/70	L.P.	4	22	3/200	L.P.			Plus 6 mos. routine

Abbreviations: DMV = Diffuse meningovascular; GP = General paresis; ND = Not determined; I.S. = Intraspinal; I.C. = Intracisternal; H.M. = Hand movements only; L.P. = Light perception only.

TABLE 10

DATA ON 11 PATIENTS WITH OPTIC ATROPHY TREATED INITIALLY WITH MALARIA THERAPY, WITH OR WITHOUT ROUTINE TREATMENT

History Number	Diagnosis	Duration Optic Atrophy at Start of Malaria (months)	Visual Acuity at Start of Treatment		Observation After Malaria (months)	Total Observation Period (months)	Final Visual Acuity		Type and Degree of Visual-Field Changes Before and After Treatment When Known		Remarks
			Better eye	Worse eye			Better eye	Worse eye	Before	After	
1) W.R.	GP	No sympsoms	20/15	20/15	96	96	20/15	20/15	I Slight	Slightly worse	Optic atrophy discovered by routine examination in midst of treatment for GP. Duration of field defect unknown
2) U 24054	GP	No symptoms	20/15	20/15	140	140	20/20	20/20	I Moderate	Normal	Plus 3½ yrs. routine Rx
3) 180819	GP	2	20/70	20/100	63	65	20/70	20/100	IV Moderate	Worse	Plus 1 yr. routine Rx
4) J 29407	Tabes	5	15/200	15/200	19	24	0	0			
5) 180820	GP	9	20/100	2/200	37	46	20/200	3/200	ND	IV Moderate	Plus 2½ yrs. routine Rx
6) U 49599	Tabes	12	20/40	0	90	102	20/200	0	IV Advanced	No change	Plus 1½ yrs. routine Rx. Central scotoma appeared during malaria and persisted
7) K 77938	DMV	10	20/40	20/200	12	22	20/70	0	II Moderate	Worse	Plus 6 mos. routine Rx
8) U 50777	GP	36	20/15	20/30	90	126	20/30	20/30	I Advanced	Same	Plus 2 yrs. routine Rx
9) 148998	DMV	15	20/100	20/100	46	61	20/100	20/200	Normal	I Moderate	4 Neo.; 6 Bi; 13 Tryp. after original observation and diagnosis. Constriction of fields noted during tryparsamide therapy, which was stopped
10) 166331	DMV	5	20/15	20/40	19	24	20/15	20/100	IV Moderate	No change	Plus 44 mapharsen; 30 Bi.
11) 158613	Cong.	No symptoms	20/30	20/50	24	24	20/30	20/70	II Moderate	No change	Plus 6 Bi.

Abbreviations: GP = General paresis; DMV = Diffuse meningovascular; Cong. = Congenital.

TABLE 11

DATA ON 39 PATIENTS WITH OPTIC ATROPHY TREATED BY A COMBINATION OF METHODS

Number		Diagnosis	Interval from Onset of Symptoms to Start of Treatment (months)	Visual Acuity Before Treatment		Initial Treatment	Summary Routine Treatment: Adequate or Inadequate	Period Observation After Initial Treatment (months)
				Better eye	Worse eye			
1	U 49716	Tabes	24	ND	ND	Routine 8 mos.	Adequate	8
2	U 29437	GP	No symptoms	20/15	20/15	Routine 3 yrs.	Adequate	37
3	M E	DMV	24	ND	ND	Routine 1 yr.	Adequate	12
4	132794	DMV	9	20/40	20/40	Routine 1 yr.	Adequate	24
5	M.G.	DMV	1	20/15	15/200	Routine 1 yr.	Adequate	12
6	U 37199	GP	1	20/40	C.F.	Routine 10 mos.	Adequate	10
7	U 54841	DMV	1	20/15	20/15	Routine 6 mos.	Adequate	6
8	U 14953	Cong.	1	ND	ND	Routine As. ×15	Adequate	4
9	H.C.	Tabes	ND	ND	ND	Routine 4 yrs.	Adequate	60
10	E.K.	DMV	3	20/20	20/30	Routine 100 As.; 200 Bi.	Adequate	108
11	U 35662	GP	6	20/20	5/200	Routine 8 mos.	Adequate	8
12	U 64620	Tabes	ND	20/20	ND	Routine 20 As.; 20 Bi. elsewhere	Adequate	102
13	U 14000	Tabes	16	20/40	5/200	Routine 19 As.; 4 Bi.	Adequate	19
14	J.S.	DMV	2	ND	ND	Routine 4 As.; 24 Bi.	Adequate	8
15	U 18808	DMV	8	20/80	20/80	Routine 3½ yrs. irreg.	Adequate	49
16	U 15729	Tabes	4	20/20	ND	Routine 6 mos.	Adequate	6
17	U 35993	Tabes	36	20/15	20/100	Routine 3 yrs. irreg.	Adequate	36
18	R.A.	Tabes	ND	ND	ND	Routine 1 yr. plus 13 diathermy treatments	Adequate	15
19	194808	DMV	ND	ND	ND	Routine 1½ yrs.	Adequate	21
20	U 13615	DMV	No symptoms	6/9	6/24	Routine 9 mos. plus 2 tryparsamide	Adequate	9
21	U 32855	DMV	No symptoms	20/20	20/100	Routine 26 As. plus mercury	Adequate	24
22	U 38985	GP	1	ND	ND	Routine 20 As. plus mercury	Adequate	11
23	U 36698	Cong.	No symptoms	20/20	20/20	Routine 3 mos.	Inadequate	3
24	191466	DMV	1	20/40	20/50	Routine 10 As.; 5 Bi.	Inadequate	4
25	T.G.	GP	2	20/20	20/20	Routine 6 As.	Inadequate	57
26	U 7257	DMV	14	20/20	8/200	Routine 7 As.	Inadequate	2
27	137730	DMV	2	20/20	20/200	Routine 10 As.	Inadequate	60
28	G.L.	Tabes	3	ND	ND	Routine mercury	Inadequate	20
29	113591	GP	No symptoms	20/15	20/15	Routine As. 11; Bi. 2	Inadequate	4
30	216220	Cong.	ND	ND	ND	Routine As. 10	Inadequate	5
31	157928	Tabes	ND	ND	ND	Routine 4 As.; 4 Bi.	Inadequate	2
32	133049	DMV	No symptoms	20/15	20/15	6 I.S.		11
33	L.M.	DMV	2	20/15	10/200	10 I.S.		18
34	U 50152	DMV	3	20/50	20/100	4 I.S.		6
35	150447	Tabes	5	5/200	5/200	2 I.C.		1
36	H 12092	Tabes	8	20/20	20/30	5 I.S.		35
37	U 11226	Tp	9	20/30	2/200	5 I.S.		2
38	131769	Tabes	4	20/70	20/70	Malaria		6
39	F.P.	DMV	3	20/20	20/20	Malaria		3

Abbreviations: GP = General paresis; DMV = Diffuse meningovascular; Cong. = Congenital; Tp = Taboparesis; ND = Not determined; C.F. = Counts fingers at five feet; H.M. = Hand movements only; L.P. = Light perception only; As. = Arsenical; I.S. = Intraspinal; I.C. = Intracisternal.

TABLE 11—*Continued*

DATA ON 39 PATIENTS WITH OPTIC ATROPHY TREATED BY A COMBINATION OF METHODS

Number		Visual Acuity		Second Type of Treatment	Period Observation After 2d Type Rx (months)	Visual Acuity After 2d Type Rx		Third Type of Treatment	Period Observation After 3d Type Rx (months)	Visual Acuity After 3d Type Treatment	
		Better eye	Worse eye			Better eye	Worse eye			Better eye	Worse eye
1	U 49716	>20/200	>20/200	3 I.S.	6	20/200	20/200				
2	U 29437	20/15	20/15	Malaria	132	20/15	20/15				
3	M.E.	20/30	L.P.	Malaria	11	20/70	0				
4	132794	20/40	20/50	5 I.C.	26	20/70 Uncor	20/200 rected	Malaria	84	20/70 Uncor	20/200 rected
5	M.G.	20/150	2/200	16 I.S.	187	10/200	2/200				
6	U 37199	20/70	0	Malaria	6	20/70	0	4 I.C.	102	15/200	0
7	U 54841	20/15	20/15	Malaria	29	20/15	20/15				
8	U 14953	20/200	2/200	6 I.S.	25	L.P.	L.P.				
9	H.C.	20/200	4/100	Malaria	16	11/200	H.M.	3 I.S.	19	11/200	H.M.
10	E.K.	20/30	20/40	12 I.C.	49	20/30	20/70				
11	U 35662	20/20	5/200	Malaria	1	20/20	5/200	7 I.C.	115	20/20	H.M.
12	U 64620	20/20	0	5 I.S.	7	20/15	0	Malaria	56	20/15	0
13	U 14000	20/60	C.F.	7 I.S.	56	L.P.	0				
14	J.S.	20/100	20/300	4 I.S.	2	8/200	H.M.				
15	U 18808	10/200	0	3 I.C.	54	H.M.	0				
16	U 15729	20/20	20/100	Malaria	41	20/20	20/100	10 I.C.	117	20/30	20/200
17	U 35993	20/15	20/100	6 I.S.	48	20/20	20/200				
18	R.A.	12/200	3/200	Malaria	48	12/200	3/200				
19	194808	>20/30	>20/100	Malaria	19	20/30	20/100				
20	U 13615	6/15	6/36	Malaria	160	20/200	20/200				
21	U 32855	20/20	20/70	2 I.S.	1	20/20	20/70	Malaria	7	20/20	20/70
22	U 38985	20/60	H.M.	2 I.S.	4	14/200	H.M.				
23	U 36698	20/40	20/50	12 I.C.	117	20/70	20/70				
24	191466	20/70	20/200	Malaria	74	20/70	20/100				
25	T.G.	20/60	20/80	Malaria	114	20/100	5/100				
26	U 7257	20/200	H.M.	5 I.S.	11	4/200	L.P.				
27	137730	20/30	C.F.	20 I.S.	228	15/200	8/200				
28	G.L.	20/15	0	Malaria	21	20/15	0				
29	113591	20/15	20/15	Malaria	36	20/15	20/15				
30	216220	20/100	C.F.	Malaria	43	20/20	20/50				
31	157928	20/70	0	Malaria	24	Useful vision for laborer					
32	133049	20/15	20/15	Malaria	48	20/15	20/15				
33	L.M.	20/15	2/200	Malaria	123	20/15	L.P.				
34	U 50152	18/200	9/200	Malaria	26	4/200	4/200				
35	150447	5/200	5/200	Malaria	108	12/200	5/200				
36	H 12092	20/30	0	Malaria	42	20/30	0				
37	U 11226	16/200	2/200	Malaria	1	0	0				
38	131769	20/200	20/200	3 I.C.	52	5/200	3/200				
39	F.P.	20/50	L.P.	1 I.S.	½	H.M	L.P.				

TABLE 11 (*Continued*)

DATA ON 39 PATIENTS WITH OPTIC ATROPHY TREATED BY A COMBINATION OF METHODS

Number		Total Period Observation (months)	Type and Degree of Visual-Field Changes		Remarks
			Before treatment	After treatment	
1	U 49716	38			I.S. followed by 14 mos. routine treatment
2	U 29437	169	II Slight	ND	Final visual acuity based on letter from patient: "Vision excellent"
3	M.E.	47	III Moderate	No change	
4	132794	143	IV Slight	No change	
5	M.G.	200	IV Advanced	No change	
6	U 37199	119	II Advanced	Worse	
7	U 54841	36	I Slight	I Moderate	Field worse on right; same on left
8	U 14953	30			
9	H.C.	95	ND	II Moderate	
10	E.K.	160			
11	U 35662	130	IV Advanced	Worse	I.C. followed by 2 yrs. routine treatment
12	U 64620	165	I Moderate (before I.S.)	No change	
13	U 14000	91			I.S. followed by 1½ yrs. routine treatment
14	J.S.	12	ND	III Advanced	
15	U 18808	111	II Advanced	Worse	
16	U 15729	168	IV Slight	No change	
17	U 35993	120	II Slight	No change	
18	R.A.	63	IV Advanced	No change	Final visual acuity based on physician's report: "Eyes unchanged"
19	194808	40	ND	III Moderate	Malaria followed by 19 mos. routine treatment
20	U 13615	169			Malaria followed by 4 mos. routine treatment and then 12 doses bismuth each year
21	U 32855	32	III Moderate (before I.S.)	Unchanged	
22	U 38985	16	ND	IV Advanced	
23	U 36698	120	III Slight	Sl. worse	I.C. followed by 6 mos. routine treatment
24	191466	79	IV Advanced	No change	
25	T.G.	173	I Slight	I Moderate	Malaria followed by 20 doses bismuth
26	U 7257	27	II Advanced	No change	
27	137730	290	IV Advanced	No change	
28	G.L.	44			
29	113591	40	II Slight	No change	Malaria followed by 1½ yrs. routine treatment
30	216220	48	I Advanced	I Moderate	Malaria followed by 2 yrs. routine treatment
31	157928	26	IV Advanced	ND	Malaria followed by 6 mos. routine treatment
32	133049	59	II Slight	Normal	Malaria followed by 1½ yrs. routine Rx and 1 yr. tryp.
33	L.M.	143	ND	I Slight (rt.) 0 (left)	Malaria followed by 2½ yrs. routine treatment and tryp.
34	U 50152	35	IV Moderate	Worse	
35	150447	114	III Advanced	No change	Malaria followed by 2 yrs. routine treatment
36	H 12092	85	I Slight	Worse	
37	U 11226	12			
38	131769	62	IV Moderate	Worse	I.C. followed by 2 yrs. routine treatment
39	F.P.	6½	II Moderate	0	Blindness precipitated by subdural treatment

untreated patients, the fact that this criterion is not an ideal one introduces no serious error.

Method of study

Since none of the patients in our study, or in any other so far reported in the literature, has been under continuous objective ophthalmologic observation from the start of infection with syphilis until the development of blindness from optic atrophy, there is no absolutely accurate method of dating the onset of optic atrophy. The only method applicable to all patients, treated and untreated alike, is to date the onset at the first appearance of visual failure noted by the patient; that is, at onset of symptoms. In the absence of symptoms the onset is dated at the time of initial ophthalmologic diagnosis based on study of fields, fundi, and visual acuity.

Having defined blindness as vision of 10/200 or less in the better eye and having determined arbitrarily to date the onset of optic atrophy at the onset of symptoms, we have now to set up criteria for the determination of the success or failure of any given type of treatment. Only treatment given subsequent to the onset of optic atrophy is considered. Since no form of treatment will restore vision already lost, patients blind before treatment are not used in the evaluation of treatment. The most that can be expected of any method of therapy is arrest of progress of visual failure. The primary desideratum, therefore, is to determine for untreated patients the cumulative percentage of blindness with passage of time dating from onset of symptoms, and to compare with this the cumulative percentage of blindness with passage of time for patients given various types of treatment, likewise dating from onset of symptoms. If the two groups of patients are comparable, and if there is a significant difference in cumulative percentage of blindness between treated and untreated patients, treatment may be adjudged to have been responsible for this difference, and hence may be termed "successful." If, on the other hand, there is no significant difference in cumulative percentage of blindness, treatment may be termed a "failure." If the treated patients represent a selected group of slow progressors, extreme caution is necessary in attributing to treatment alone any observed difference in cumulative percentage of patients blind.

In order to determine the cumulative percentage (probability) of blindness, it is first necessary to ascertain the annual rate of blindness for each year after onset of symptoms. From this the "probability of blindness" within a given year and the cumulative probability of blindness can be calculated (Reed and Merrell[9]). The annual "blind" rate is calculated by dividing the number of patients who go blind during a given year by the average number of patients under observation (that is, "at risk") during that particular year. In order to assemble the data in a form that takes account of the individual variation in observation period, an adaptation of the conventional life table[10] is used. The unit is one person under observation for one year, or one person-year.

Derivation of the life tables

The life tables are derived from the basic data presented in tables 7 to 11, inclusive. Since the unit of a life table is a person-year rather than a person, it is possible to utilize some patients twice, or, in some instances, even three times by making up life tables in which each patient is entered only for that period after onset of symptoms during which he was "at risk" of going blind following a particular type of treatment. To illustrate the method: A patient was given malaria

in January, 1937. When first seen at that time he had visual acuity of 20/100 and a history of onset of symptoms in March, 1936. At the time of last observation (February, 1940) his vision was 20/200 and he was still "not blind." Though this patient's total course from onset of symptoms to final observation represents a period of 46 months, he was evaluated in the "no treatment" group for that portion of his course before treatment was given (9 months), and in the malaria-treated group only for that portion of his course subsequent to treatment (37 months).

Of the 250 patients comprising this study, 131 were evaluated only once, 94 were evaluated twice, and 25 were evaluated three times. The sum of the life tables is therefore 394 "persons." The total number of person-years is 836.

Critique of the material

In the evaluation of any method of treatment a control group of untreated patients is essential. Ideally, moreover, this control group and the treated group should be the same in all relevant respects. This is possible, in planned experiments, by the alternate assignment of patients to the treated and untreated groups. In a retrospective study of the present type no such control group is available, and the course of the untreated disease must be estimated almost entirely from purely anamnestic data. Patients who received no treatment at any time were, for the most part, those who were blind or almost blind at the time of our first observation.

Data based on patients' histories are notoriously unreliable. This is particularly true of a process such as primary optic atrophy. The disease must *a priori* have progressed to a greater or less degree before the patient will have noted visual failure. The duration of symptoms as related to the examiner will be influenced by several extraneous factors. Not the least of these are the care with which the history is taken and the accuracy of the patient's memory. In the analysis of individual case records it was repeatedly noted that the elicited onset of symptoms varied from examiner to examiner. A second extraneous factor is the intelligence of the patient, his occupation, and the accuracy of his powers of observation. Even the most intelligent may proceed about his routine tasks oblivious of visual difficulty until a perhaps fortuitous refraction and prescription of glasses reveals a bright new world. A third factor influencing duration of symptoms is the type of visual-field defect. The patient with a central scotoma will obviously become aware of his disability earlier in the course of his disease than will the patient with only concentric constriction of the peripheral fields.

It is necessary further to inquire whether the treated patients represent a selected group. This problem is best approached by comparison of the rate of progress of visual failure *prior to* treatment in this group with the rate of progress of visual failure in the untreated group. Such a comparison is valid since extraneous factors influencing the apparent duration of symptoms are common to all patients. Figure 1 is a scatter chart in which the results of this comparison are graphically depicted. In 15 of the treated patients therapy was begun early in the course of the disease before onset of symptoms. It is therefore impossible to predict their course had they remained untreated. There were, however, 76 patients who had had symptoms for variable but stated periods of time before the institution of treatment and in whom the visual acuity before treatment had been objectively measured. Inspection of the scatter chart re-

veals that in these patients visual acuity was better maintained even without treatment than in the control group. The mean visual acuity in the two groups relative to duration of symptoms can be derived from this scatter chart. This is facts that the course of untreated optic atrophy had of necessity to be estimated from anamnestic data influenced by a multiplicity of extraneous factors and that the treated patients, particularly the malaria and subdural series, represent a

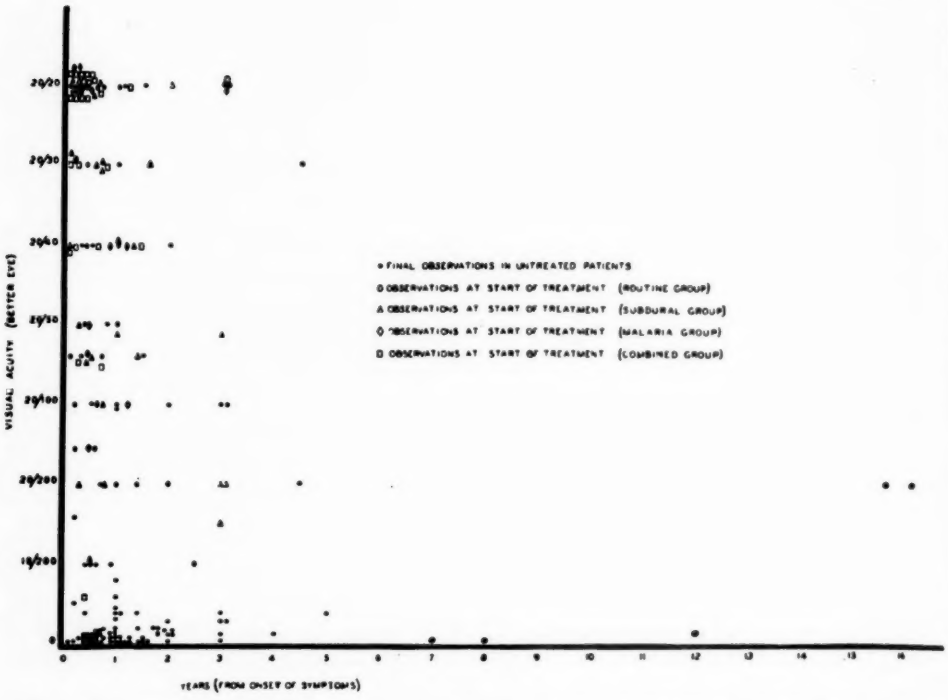
VISUAL ACUITY (BETTER EYE)
YEARS (FROM ONSET OF SYMPTOMS)
• FINAL OBSERVATIONS IN UNTREATED PATIENTS
O OBSERVATIONS AT START OF TREATMENT (ROUTINE GROUP)
Δ OBSERVATIONS AT START OF TREATMENT (SUBDURAL GROUP)
◊ OBSERVATIONS AT START OF TREATMENT (MALARIA GROUP)
□ OBSERVATIONS AT START OF TREATMENT (COMBINED GROUP)

Fig. 1 (Moore *et al.*). Relationship between duration of symptoms and visual acuity in untreated optic atrophy: Scatter chart demonstrating that the treated patients are a selected group.

Each observation represents an individual patient. Abscissa indicates elapsed time between onset of symptoms and institution of treatment (in group later treated). In untreated group it indicates elapsed time between onset of symptoms and final observation. Ordinate indicates visual acuity in better eye.

done in figure 2. In this chart, for reasons which will be apparent later, patients subsequently given only routine treatment are included with those never treated (curve A) for comparison with patients subsequently given malaria or subdural therapy (curve B). It is apparent that the latter represent a selected group in whom the process proceeds toward blindness relatively slowly, even in the absence of treatment.

Bearing in mind the two important selected group of slow progressors, we may now proceed to an analysis of the course of the untreated disease and the influence of various types of treatment upon its progress.

The course of untreated optic atrophy

Subject to the limitations discussed under critique of material, our data permit, for the first time, an estimation of the progress of untreated primary optic

atrophy. Were this estimation to be based alone on patients who were never treated, a falsely gloomy impression would result. In figures 1 and 2 we have demonstrated that these patients represent the most rapid progressors. In order partially to obviate this difficulty, the data on this group of 89 patients are augmented by data on 108 patients subsequently treated, utilizing only that portion of their course prior to treatment. By this device we partially compensate for selection of material, but only in the sense that the resultant picture of the untreated disease is a composite one derived in part from the slow progressors (subsequently treated) and in part from the rapid progressors (never treated). Any observed difference between untreated and treated groups is therefore of greater significance than if we had utilized as a control group only those patients who were never treated.

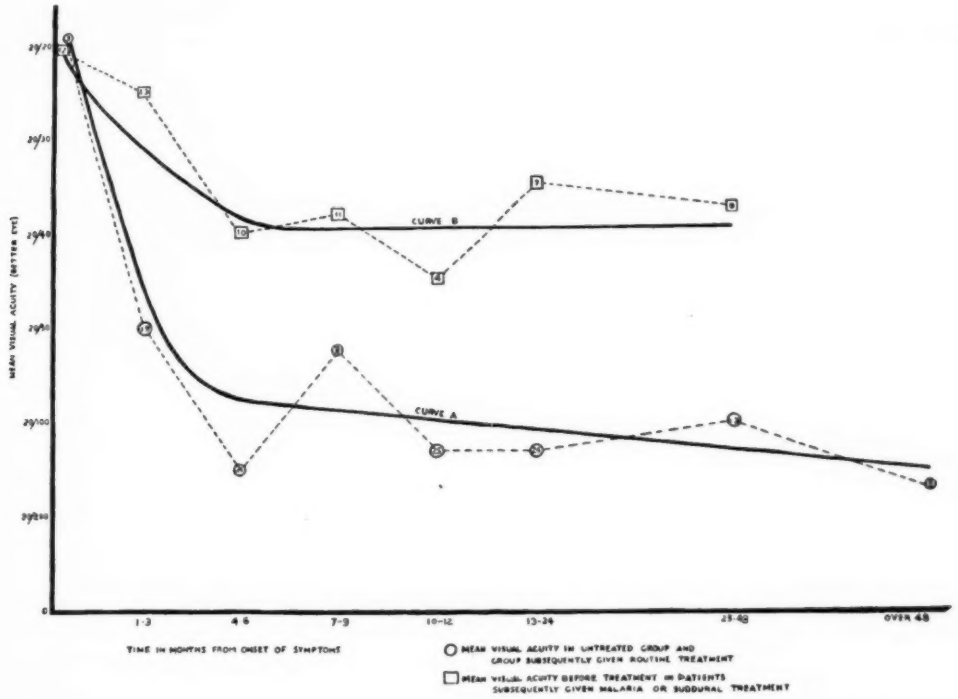

Fig. 2 (Moore *et al.*). Mean visual acuity in untreated optic atrophy relative to duration of symptoms, demonstrating that patients subsequently treated with malaria and/or subdural treatment are a selected group.

Abscissa and ordinate are as in figure 1. The number of patients upon which each observation is based is indicated by the appropriate numeral. The actual observations are connected by dotted lines through which the curves are smoothed by hand. Curve A represents mean visual acuity of patients never treated and those subsequently given only routine treatment. Curve B represents mean visual acuity *before treatment* of patients subsequently given malaria and/or subdural therapy.

Table 12 shows in detail the summation of life experience in untreated optic atrophy. It is based on 252.5 person-years of observation, for the most part retrospective. The cq_x column of table 12 is presented in graphic form in curve A of figure 3, in which the abscissa repre-

sents the time in years dating from onset of symptoms and the ordinate represents the cumulative probability of blindness. We find that blindness may be expected to occur in 28 percent of untreated patients within one year of onset of symptoms, in 50 percent within two years, and in almost 65 percent within three years. Not until six years after onset of symptoms are 75 percent of the patients blind. Particular attention is called to this steep slope of the curve at the onset, and its subsequent flattening, since this configuration is most significant for the proper evaluation of treatment. Succinctly expressed, we have approximately 65 percent of patients blind 3 years after onset of symptoms, whereas after 12 years only an additional 20 percent are blind. This indicates that patients not blind at the end of three years subsequently did quite well, even in the absence of treatment. Hence in patients who have had symptoms for three or more years before the institution of treatment it is not valid to attribute a subsequent favorable course to treatment.

TABLE 12

THE CUMULATIVE PERCENTAGE OF BLINDNESS IN UNTREATED SYPHILITIC PRIMARY OPTIC ATROPHY
DATA BASED ON 252.5 PERSON-YEARS

Years After Onset of Symptoms	Time* Period	l_x†	w_x	b_x	L_x	100 M_x	100 Q_x	P_x	CP_x	CQ_x
0–1	X1	197	88	43	131.5	32.7	28.1	71.9	71.9	28.1
1–2	X2	66	18	17	48.5	35.1	29.9	70.1	50.4	49.6
2–3	X3	31	10	7	22.5	31.1	26.9	73.1	36.9	63.1
3–4	X4	14	4	1	11.5	8.7	8.3	91.7	33.8	66.2
4–5	X5	9	2	1	7.5	13.3	12.5	87.5	29.6	70.4
5–6	X6	6	1	1	5.0	20.0	18.2	81.8	24.2	75.8
7–8	X8	4	0	1	3.5	28.6	25.0	75.0	18.3	81.7
11–12	X12	3	0	1	2.5	40.0	33.3	66.7	12.1	87.9
14–15	X15	2	1	0	1.5	0.0	0.0	100.0	12.1	87.9
16–17	X17	1	1	0	0.5	0.0	0.0	100.0	12.1	87.9

* In the above and subsequent life tables, all time intervals date from onset of symptoms.
† The following definitions and formulas apply to all the life tables:

Primary figures

Time period X = 1 year (unless otherwise specified)
l_x = Number of patients at beginning of time period$_x$
a_x = Number of patients added during time period$_x$ (used only in subsequent tables having to do with treated patients)
w_x = Number of patients lost from observation during time period$_x$, not blind
b_x = Number of patients blind during time period$_x$

Derivatives from primary figures

L_x = Average number of patients observed during time period$_x$ = $l_x + \frac{1}{2}a_x - \frac{1}{2}w_x - \frac{1}{2}b_x$

M_x = Blind rate during time period$_x$ = $\frac{b_x}{L_x}$

Q_x = Probability of blindness during time period$_x$ = $\frac{2M_x}{2+M_x}$ (Reed and Merrell)[9]

The actual figure used is 100 Q_x which is the percentage of patients blind during period.
P_x = Percentage of patients not blind during time period$_x$ = 100 − 100 Q_x
CP_x = Cumulative percentage of patients not blind = $P_{x1} \cdot P_{xn}$
CQ_x = Cumulative percentage of patients blind = 100 − CP_x

ROUTINE TREATMENT

The basic data for patients given routine treatment alone are shown in table 8. To the 79 patients of table 8

have been added 31 others from table 11 (treated by combined methods), whose treatment was begun by the routine system. These latter 31 are here considered only in so far as the effect of routine treatment is concerned, being withdrawn from the life table when a special form of therapy (subdural or malaria) was administered. Of the total 110 patients, 46 received less than 10 injections of a trivalent arsenic and 10 injections of a heavy metal, and 64 received more than 10 injections of each. These two groups of patients are at first to be considered separately.

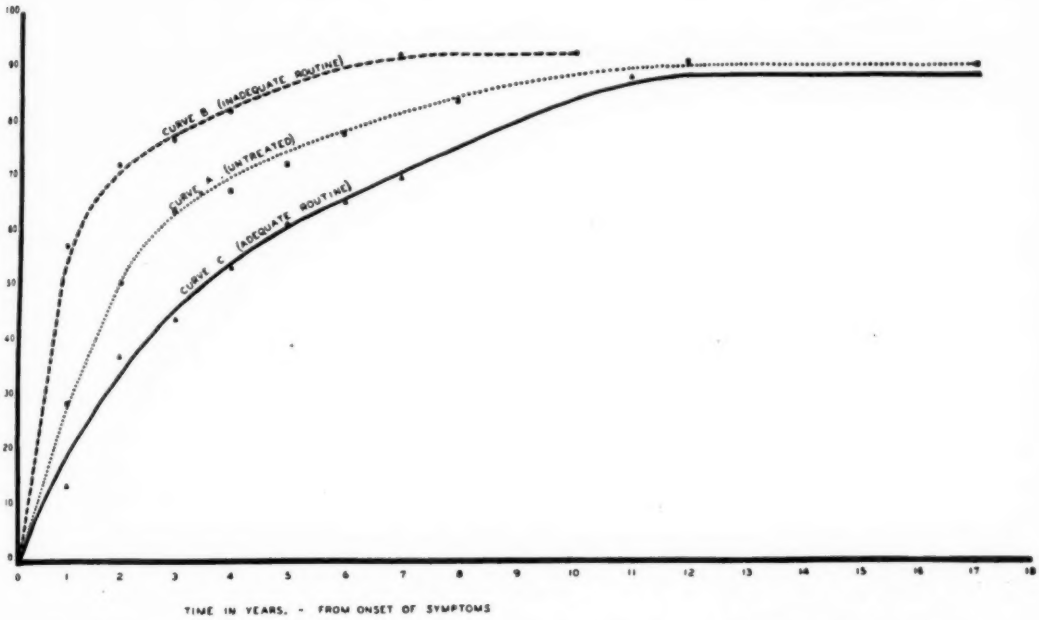
CURVE B (INADEQUATE ROUTINE)
CURVE A (UNTREATED)
CURVE C (ADEQUATE ROUTINE)
CUMULATIVE PERCENTAGE OF BLINDNESS
TIME IN YEARS. - FROM ONSET OF SYMPTOMS

Fig. 3 (Moore *et al.*). The cumulative percentage of blindness in untreated syphilitic primary optic atrophy (curve A) as compared with the cumulative percentage of blindness under inadequate routine treatment (curve B) and under adequate routine treatment (curve C). In this and subsequent graphs, the curves are smoothed by hand.

Inadequate treatment. Due to the fact that most of these 46 patients were treated elsewhere, the data are in many cases incomplete. For 18 patients of this group, we do not know at what time period after onset of symptoms treatment was first given. It is therefore impossible to enter them in the appropriate a_x column of the life table and they are arbitrarily included in the lx_1 group of patients treated at onset of symptoms. As an example we may cite a patient already blind at the time of our first observation who gave a history of onset of symptoms 14 months previously, with inadequate treatment administered at an unstated period after onset of symptoms. This patient, for want of more precise data, was considered to have maintained vision for 14 months after the institution of treatment, whereas actually the time period after treatment might have been only 12, 10, or even 6 months. Since there were 18 such patients, constituting over one third of the entire group, it is evident that, by this device, the data as presented are weighted

in favor of treatment. Therefore if, even under these circumstances, inadequate routine treatment is shown to be of no value, we may be quite certain of our facts.

That such is precisely the case is shown in table 13, which presents in detail the summation of life experience in the inadequate routine treatment of primary optic atrophy. It is based upon only 55 person-years of observation. The cumulative percentage of blindness is presented in graphic form in curve B of figure 3.

TABLE 13

CUMULATIVE PERCENTAGE OF BLINDNESS IN SYPHILITIC PRIMARY OPTIC ATROPHY TREATED INADEQUATELY BY ROUTINE METHODS. DATA BASED ON 55.0 PERSON-YEARS

Years After Onset of Symptoms	Time Period	l_x	a_x	w_x	b_x	L_x	100 M_x	100 Q_x	P_x	CP_x	CQ_x
0–1	X1	24	21	9	17	21.5	79.1	56.8	43.2	43.2	56.8
1–2	X2	19	1	7	6	13.0	46.2	34.9	65.1	28.1	71.9
2–3	X3	7	—	1	1	6.0	16.7	15.4	84.6	23.8	76.2
3–4	X4	5	—	0	1	4.5	22.2	19.9	80.1	19.0	81.0
4–5	X5	4	—	1	0	3.5	0.0	0.0	100.0	19.0	81.0
5–6	X6	3	—	1	0	2.5	0.0	0.0	100.0	19.0	81.0
6–7	X7	2	—	0	1	1.5	66.7	50.0	50.0	9.5	90.5
9–10	X10	1	—	1	0	0.5	0.0	0.0	100.0	9.5	90.5

This curve has the same configuration as the curve for untreated patients (curve A) with a steep slope at the onset and subsequent flattening. The probability of blindness at any time period after onset of symptoms is, however, greater than in the untreated group—approximately 57 percent as contrasted with 28 percent in one year, 72 percent as contrasted with 50 percent in two years, and 76 percent as contrasted with 63 percent in three years. Thereafter the curves approach each other more closely but never approximate.

There are two possible explanations for the obvious fact that patients given inadequate routine therapy did worse than patients who received no treatment at all. Either the treatment itself was deleterious, or the treated patients represent a selected group of rapid progressors. A study of the basic data for individual patients (table 8) reveals that the latter explanation is the correct one. Nine patients were blind in less than six months after onset of symptoms and two additional patients were blind less than six months after treatment was begun. Thus 11 of the 46 patients, almost 25 percent, did not retain vision long enough to get the minimal amount of treatment here defined as adequate; that is, they were blind literally before they had time to get adequate treatment. It is this group of patients with rapidly progressing optic atrophy which accounts for the difference between curves A and B of figure 3. It is apparent that the arbitrary subdivision of patients given routine treatment into those treated inadequately and those treated adequately is an artificial one, and what we have actually done is to divide this group into two subgroups of relatively rapid and slow progressors. The validity of this statement is further borne out by facts subsequently to be presented.

Adequate treatment. The actual life table for this group is omitted. The cq_x column (cumulative percentage of blindness), derived in the same manner as in the preceding life tables, is shown in column C of table 14. For 14 of the 64 patients given adequate routine treatment, we do not know at what period after onset of symptoms this treatment was first

given, and they are arbitrarily included in the lx_1 group of patients treated at onset of symptoms. The data as presented are thus weighted in favor of treatment. The cumulative percentage of blindness is presented in graphic form in curve C of figure 3. This curve has the same configuration as the curve for inadequately treated patients (curve B) and the curve for untreated patients (curve A). The probability of blindness at any time period after onset of symptoms is, however, less than in the untreated group—approximately 13 percent as contrasted with 28 percent in one year, 37 percent as contrasted with 50 percent in two years, and 43 percent as contrasted with 63 percent in three years. Thereafter the two curves approach each other and approximate at the eleventh year.

At first glance it would seem that adequate routine therapy might possibly be of some slight value in delaying the progress of optic atrophy, even though by the eleventh year the percentage of patients blind in both the untreated and the treated groups is approximately the same. The following facts, however, preclude any such conclusion. First, the data as presented are weighted in favor of the treated group because of inadequate data in 14 of the 64 patients. Second, in the arbitrary subdivision of patients routinely treated, we have already artificially selected the most rapid progressors for consideration under the subgroup of patients inadequately treated. It follows as a corollary that the remainder (those adequately treated) represent the subgroup with the better initial prognosis, and that

TABLE 14

THE CUMULATIVE PERCENTAGE OF BLINDNESS IN SYPHILITIC PRIMARY OPTIC ATROPHY

	A	B	C	D(B+C)	E(A+D)	F	G
Years After Onset of Symptoms	Untreated (252.5 person-years)	Inadequate Routine (55 person-years)	Adequate Routine (151 person-years)	All Routine (206 person-years)	Total Control Group (458.5 person-years)	Subdural (249 person-years)	Malaria (128.5 person-years)
1	28.1	56.8	13.1	34.7	30.0	40.0	8.7
2	49.6	71.9	36.7	54.5	51.0	49.8	13.5
3	63.1	76.2	43.3	60.2	60.4	55.6	18.3
4	66.2	81.0	52.4	67.0	66.0	57.4	18.3
5	70.4	81.0	60.2	70.8	70.0	61.0	18.3
6	75.8	81.0	64.0	73.0	73.2	66.4	18.3
7	75.8	90.5	68.5	78.4	77.0	66.4	18.3
8	81.7	90.5	68.5	78.4	79.1	70.2	18.3
9	81.7	90.5	74.7	82.0	81.5	72.0	18.3
10	81.7	90.5	74.7	82.0	81.5	74.3	18.3
11	81.7		87.4	91.0	85.2	74.3	18.3
12	87.9		87.4	91.0	89.0	74.3	18.3
13	87.9		87.4	91.0	89.0	74.3	18.3
14	87.9		87.4	91.0	89.0	74.3	18.3
15	87.9		87.4	91.0	89.0	74.3	18.3
16	87.9		87.4	91.0	89.0	74.3	
17	87.9		87.4	91.0	89.0	79.4	
18						79.4	
19						79.4	
20						79.4	
21						79.4	
22						79.4	
23						79.4	
24						79.4	
25						79.4	

any observed difference between this group and the untreated group is most probably due to selection. Inspection of figure 3 reveals that at any given point curve A (untreated group) lies almost midway between curve B (inadequately treated group) and curve C (adequately treated group). Indeed, it would be possible arbitrarily to divide curve A into rapid and slow progressors and get curves superimposable on curves B and C. As a matter of fact, this is precisely what we have done when we artificially divide our routinely treated group into those adequately and those inadequately treated.

In order to eliminate from consideration patients blind within a year after onset of symptoms, life tables were constructed to determine the cumulative probability of blindness among those patients who were not blind one year after onset of symptoms. The actual life tables are omitted, but the cq_x columns are presented in table 15. The curves of figure 4 are based on columns A, B, and C of this table. By this device, which in part eliminates selection of cases, the cumulative percentage of blindness in the three groups approximates more closely.

As a final measure of comparison between the untreated and the routinely treated patients, the arbitrary subdivision of the latter group into those treated adequately and those treated inadequately is

TABLE 15

THE CUMULATIVE PERCENTAGE OF BLINDNESS AMONG THOSE PATIENTS WHO DID NOT BECOME BLIND DURING THE FIRST YEAR AFTER ONSET OF SYMPTOMS

Years After Onset of Symptoms	A	B	C	D(B+C)	E(A+D)	F	G
	Untreated	Inadequate Routine	Adequate Routine	All Routine*	Total Control Group	Subdural	Malaria†
2	29.9	34.9	27.1	30.3	30.0	16.3	5.3
3	48.8	45.0	35.9	39.0	43.4	25.9	10.5
4	53.0	55.8	46.2	49.4	51.5	29.0	10.5
5	58.9	55.8	53.2	55.3	57.3	35.0	10.5
6	66.4	55.8	58.5	58.8	61.9	44.2	10.5
7	66.4	77.9	63.7	67.0	67.4	44.2	10.5
8	74.8	77.9	63.7	67.0	70.3	50.4	10.5
9	74.8	77.9	70.9	72.5	73.7	53.4	10.5
10	74.8	77.9	70.9	72.5	73.7	57.2	10.5
11	74.8		85.5	86.3	78.9	57.2	10.5
12	83.2		85.5	86.3	84.4	57.2	10.5
13	83.2		85.5	86.3	84.4	57.2	10.5
14	83.2		85.5	86.3	84.4	57.2	10.5
15	83.2		85.5	86.3	84.4	57.2	10.5
16	83.2		85.5	86.3	84.4	57.2	
17	83.2		85.5	86.3	84.4	66.7	
18						66.7	
19						66.7	
20						66.7	
21						66.7	
22						66.7	
23						66.7	
24						66.7	
25						66.7	

* Not shown in graphic form because it is almost identical with curve C of figure 3 (the curve for all patients given adequate routine treatment), and does not differ significantly from curve A of figure 4 (the curve for untreated patients who did not become blind during the first year after onset of symptoms).

† Not shown in graphic form because only 1 of the 32 malaria-treated patients was blind within a year (that is, there was only one obviously rapid progressor in the group).

discarded. All routinely treated patients are considered as a unit. The actual life table for this composite group is omitted. The cumulative percentage of blindness (cq_x) is shown in column D of table 14, and in figure 5 is compared (curve D) with the cumulative probability of blindness in the untreated group (curve A). *These curves are superimposable, conclusively demonstrating that routine treatment alone has no effect upon the course of syphilitic primary optic atrophy.*

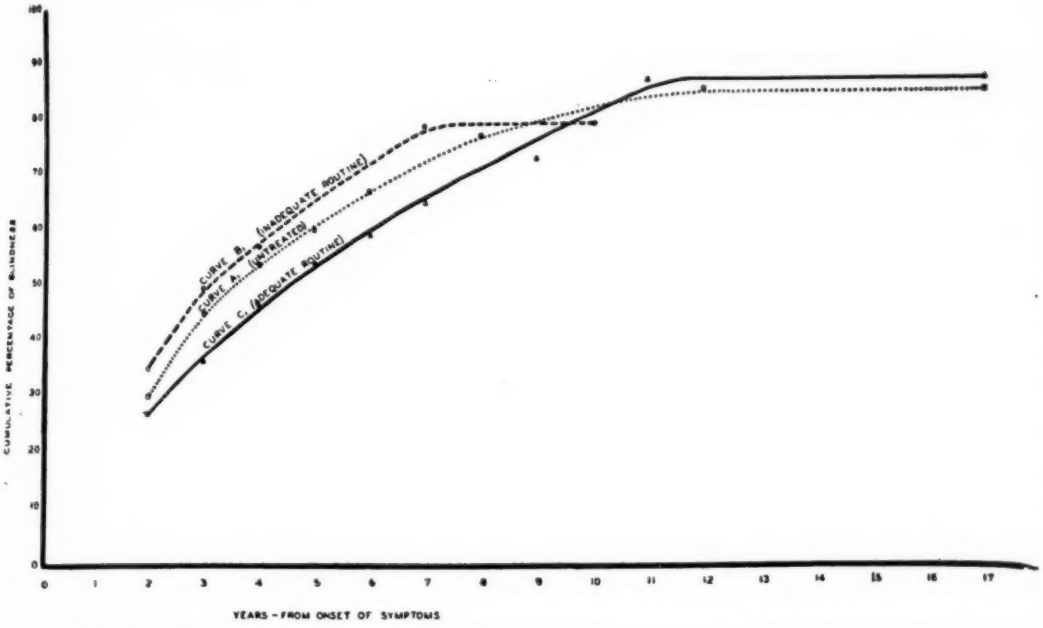
CUMULATIVE PERCENTAGE OF BLINDNESS
CURVE B, (INADEQUATE ROUTINE)
CURVE A, (UNTREATED)
CURVE C, (ADEQUATE ROUTINE)
YEARS - FROM ONSET OF SYMPTOMS

Fig. 4 (Moore *et al.*). Further comparison of untreated group (curve A_1) and routinely treated group (curves B_1 and C_1). The cumulative percentage of blindness in patients who were not blind one year after onset of symptoms. By this device, the most rapid progressors are eliminated from all groups, and the apparent differences of figure 3 tend to disappear. *There is no significant difference between the curves for untreated patients and those given either inadequate or adequate routine treatment.*

Total control group. Since the course under routine treatment is the same as the course under no treatment, the control group can be augmented by considering person-years under routine treatment as a unit with person-years under no treatment. To illustrate, if a patient remained untreated for six months after onset of symptoms and was then given only routine treatment for the ensuing six months, he may, for practical purposes, be considered as having spent 12 months without effective treatment. By this means the number of person-years of observation in the control group is raised to 458.5, an impressive total. By sheer increase in number the cumulative percentage of blindness in this control group (column E of table 14) gives a more valid picture of the course of untreated primary optic atrophy. In subsequent figures it is this group (curve E of figures 6 and 8) which is used as the standard of comparison in the evaluation of malaria and subdural therapy and is designated *control group.*

We are now in a position to clarify the

method of approach to the evaluation of treatment in the 31 patients given routine therapy followed by malaria or subdural therapy. Since it has been demonstrated that routine therapy is for practical purposes identical with no therapy, it follows that any possible good results in the group treated by combined methods must be attributed to the special forms of treatment. It is therefore permissible to evaluate the effects of fever therapy in the 16 patients of this group in whom routine treatment was followed by induced malaria, and to evaluate the effects of subdural therapy in the 15 patients in whom routine therapy was followed by the Swift-Ellis treatment.

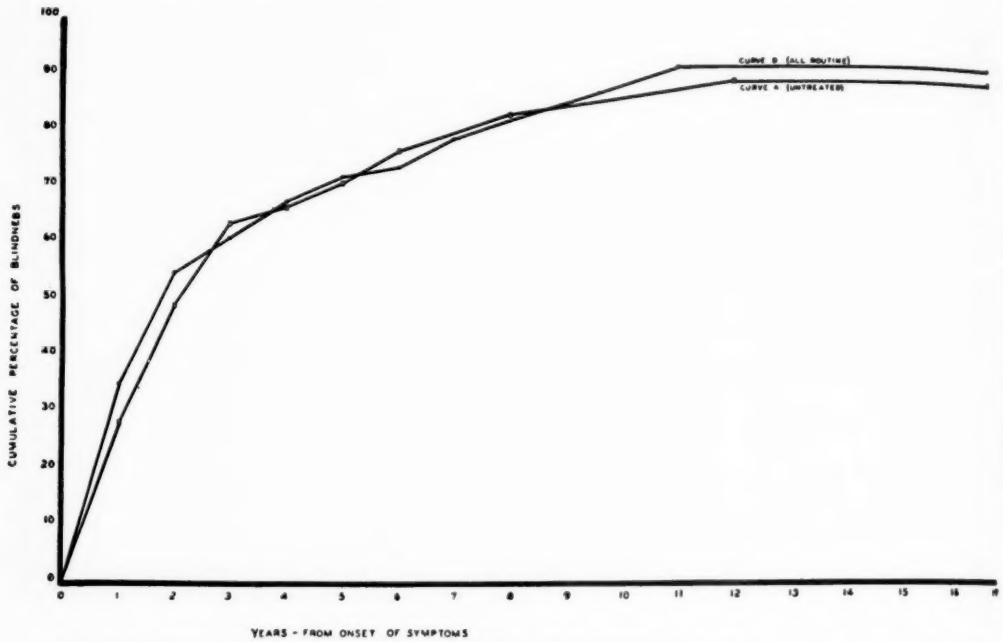

Fig. 5 (Moore *et al.*). Demonstrating that routine treatment has no effect on the progress of syphilitic primary optic atrophy. Curve D (all routinely treated patients) is superimposable upon curve A (untreated patients).

Subdural treatment

The basic data for patients whose treatment was initiated by subdural therapy are shown in table 9. In addition to these 32 patients, there are 6 others (table 11) in whom treatment was also initiated by subdural therapy which was followed at stated intervals by induced malaria. These six patients were, for obvious reasons, withdrawn from the life table at the time fever therapy was instituted. There are thus under consideration 38 patients whose treatment was initiated by subdural therapy. To these 38 patients have been added 15 others from table 11 whose treatment was initiated by more or less routine treatment which was followed by subdural therapy. These latter 15 are here considered only in so far as the effect of subdural treatment is concerned, being entered in the appropriate a_x column of the life table at the time when the first subdural treatment was given. Only 3 of these 15 patients subsequently received malaria therapy, at which time they were withdrawn from the life table.

Only six patients in the entire study received malaria followed by subdural therapy. In four of these six patients, subdural therapy could not be evaluated because the progress of the optic atrophy had already been apparently arrested by malaria. The other two patients in whom there was progression of the disease despite fever have been added to the 38 patients in whom treatment was initiated by subdural therapy and the 15 patients in whom treatment was initiated by routine therapy followed by subdural therapy.

The actual life table is omitted. The cumulative percentage of blindness (cq_x) for these 55 patients is shown in column F of table 14. It is based upon 249 person-years of observation. Thus, though the number of patients is smaller, the person-years of observation are approximately the same as in the untreated and routinely treated groups. Moreover, in contrast to the routinely treated patients, many of whom were treated elsewhere, data are available in all patients as to the duration of symptoms at the time subdural therapy was initiated and in all but two patients as to the visual acuity before treatment. Whereas the data concerning the untreated group are almost entirely anamnestic and those of the routinely treated group are in part anamnestic, the data of the subdural group are based on observation in the true sense of the word; namely, objective measurement of visual acuity before treatment and at the termination of the observation period after treatment.

The several groups of patients are

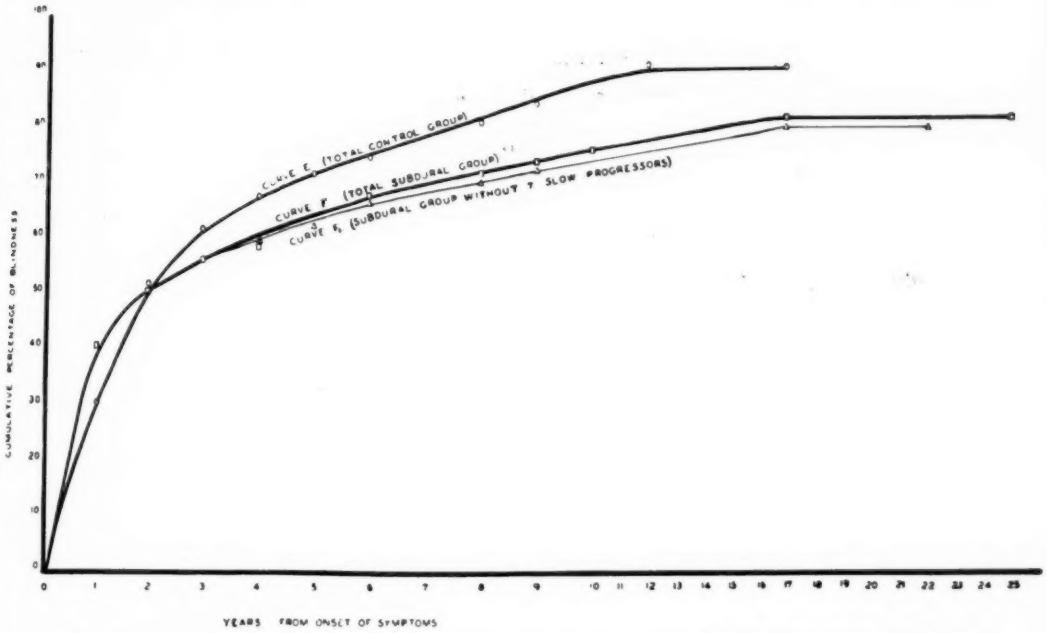
CURVE E (TOTAL CONTROL GROUP)
CURVE F (TOTAL SUBDURAL GROUP)
CURVE F1 (SUBDURAL GROUP WITHOUT 7 SLOW PROGRESSORS)
CUMULATIVE PERCENTAGE OF BLINDNESS
YEARS FROM ONSET OF SYMPTOMS

Fig. 6 (Moore *et al.*). Cumulative percentage of blindness in subdural group (curve F) compared with cumulative percentage of blindness in total control group (curve E). The apparent slight statistical advantage of subdural treatment is not decreased by elimination of the seven patients who had symptoms for more than three years before subdural therapy was instituted (curve F_1).

thus not strictly comparable. It has been shown, moreover, that in the subdural group of patients visual acuity was better maintained even without treatment than in the control group. Bearing in mind that we are dealing with a group of patients selected both as to slowness of progression of the disease and as to basic data, we may proceed to a comparison of the cumulative percentage of blindness in the patients treated subdurally with the cumulative percentage of blindness in the control group.

TABLE 16

PATIENTS IN WHOM VISUAL FAILURE WAS APPARENTLY PRECIPITATED BY SUBDURAL TREATMENT

Case Number	Diagnosis	Previous Treatment for Optic Atrophy	Duration Optic Atrophy to Start of Subdural Treatment (months)	Subdural Treatment	Visual Acuity Before Subdural Treatment		Interval to Complete Blindness (months)
					Better eye	Worse eye	
U40745	Tabes	None	5	2 I.S.	20/70	2/200	1
U23139	Tabes	None	8	5 I.S.	20/30	3/100	4
H.	General paresis	None	64	5 I.S.	20/20	0	1
U 5410	General paresis	None	No symptoms (Onset of symptoms 2 wks. *after* last intraspinal treatment)	4 I.S.	Not determined	Not determined	4
F.P.	Diffuse meningovascular	Malaria	6	1 I.S.	20/50	Light perception only	½

Figure 6 presents this comparison in graphic form. Again it is to be noted that the curve for patients given subdural treatment (curve F) has the same configuration as the curve for the control group (curve E). One is first impressed by the fact that whereas only 30 percent of the control group were blind within one year of onset of symptoms, 40 percent of patients given subdural treatment were blind within that time period. A reason for this difference is to be found in *the tendency of subdural treatment in certain instances suddenly to extinguish vision.* This occurrence has been noted in no less than 5 of the 55 patients so treated. The basic data as to these five patients, gathered in table 16, show no factor which might have permitted an anticipation of the unfortunate outcome. The fact that four of these five patients were blinded by subdural therapy within a year of onset of symptoms accounts for the higher percentage of blindness in the subdural group than in the control group during the first year. For the second and third year after onset of symptoms the cumulative percentage of blindness in the two groups is almost identical. Thereafter there is a slight but widening difference between the two curves of figure 6 so that, after 8 years 80 percent of the controls and 70 percent of the subdural group are blind, while after 12 years 90 percent of the controls and only 75 percent of the subdural group are blind.

While it is certain that any form of treatment which permits blindness in 40 percent of patients within one year of

onset of symptoms and which itself blinds almost 10 percent of patients is not highly to be recommended, sufficient evidence is present from the above data to suggest some apparent therapeutic effect, with arrest of progress of the disease in at least a small proportion of cases. Further analysis is essential to determine whether this is to be attributed to the treatment itself or is merely the result of selection of material.

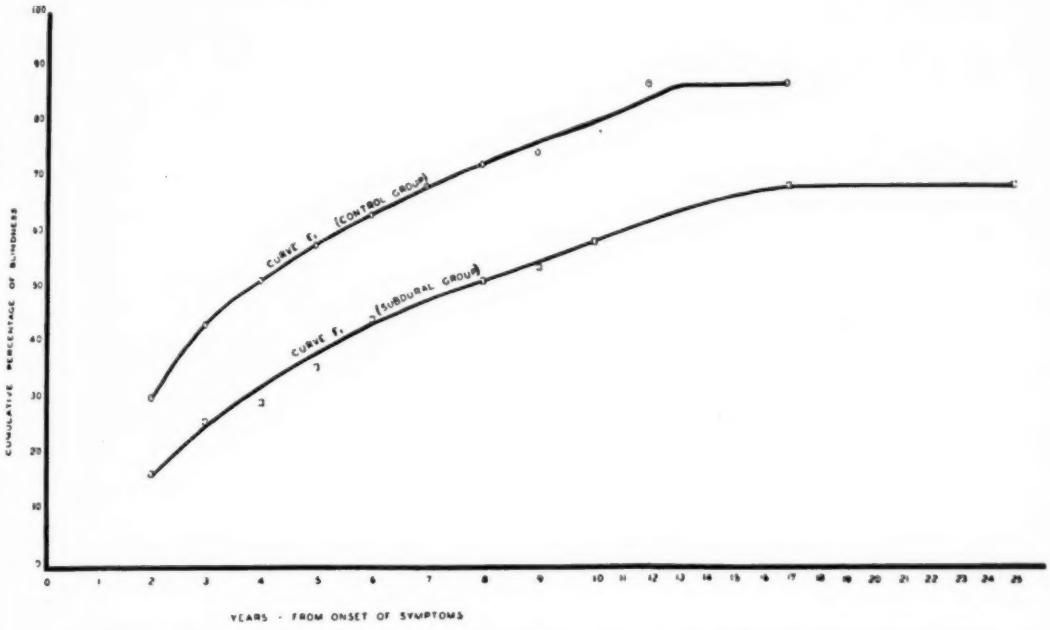

Fig. 7 (Moore *et al.*). Further comparison of subdural group (curve F_1) and total control group (curve E_1). The cumulative percentage of blindness in patients who were not blind one year after onset of symptoms. Elimination of the most rapid progressors from both groups augments somewhat the apparent slight statistical advantage of subdural therapy (compare fig. 6).

The first procedure to this end is to eliminate from consideration the most rapid progressors in both the subdural and control groups and to consider the cumulative percentage of blindness of those patients who "survived" the first year after the onset of symptoms. It is to be recalled that when the same method was applied to a comparison of the untreated and routinely treated groups (fig. 4), apparent differences tended to disappear. By this device we also eliminate from consideration four of the five patients blinded by subdural therapy. In figure 7, which is based on columns E and F of table 15, it is evident that the apparent difference between the control and the subdurally treated group is somewhat enhanced by this method of analysis. Of patients who were not blind within the first year after onset of symptoms, only 16 percent of the subdural group as compared with 30 percent of the control group were blind at the end of 2 years, only 29 percent as compared with 52 percent after 4 years, 50 percent as compared with 70 percent after 8 years, and 66 percent as compared with 85 percent 17 years after onset of symptoms.

To put the matter simply, of every 10 patients with primary optic atrophy given

subdural treatment, four will be blind within one year after onset of symptoms, one as a direct result of treatment itself. In two of the remaining six patients, the process would appear to be permanently arrested. However, one of these two patients would not have been blind even if untreated. These results are not altered by elimination of the seven patients in the group who had had symptoms more than three years before the initiation of subdural therapy (see curve F_1 of fig. 6). In view of the factors of selection previously discussed, the apparent slight statistical difference between the course of primary optic atrophy under subdural therapy and the course of the control group of patients would not appear sufficient to justify the further use of this form of treatment. Its abandonment is further justified because of the precipitation of blindness in almost 10 percent of patients so treated.

Malaria treatment

The basic data for patients whose treatment was initiated by malaria therapy are shown in table 10.* In addition to these 11 patients there were 2 others (table 11) in whom treatment was also initiated by malaria therapy which was followed at stated intervals by subdural therapy. These two patients were withdrawn from the life table at the time subdural therapy was instituted. To these 13 patients have been added 16 others from table 11 whose treatment was initiated by routine treatment which was followed by malaria. These latter 16 are here considered only in so far as the effect of malaria treatment is concerned, being entered in the appropriate a_x column of the life table at the time fever therapy was given. Only 4 of these 16 patients subsequently received subdural therapy, at which time they were withdrawn from the life table.

Only nine patients in the entire study received subdural followed by malaria therapy and in only three of these nine patients was it possible to evaluate the effects of malaria, because in the other six the process was apparently not progressive before its institution. These three patients have been added to the thirteen patients in whom treatment was initiated by malaria therapy and the sixteen patients in whom treatment was initiated by routine therapy followed by malaria therapy. The actual life table is omitted. The cumulative percentage of blindness (cq_x) for these 32 patients is shown in column G of table 14. It is based upon 128.5 person-years of observation. Figure 8 compares in graphic form the cumulative percentage of blindness in the malaria-treated patients (curve G) with the cumulative percentage of blindness in the control group (curve E). One is at once impressed with the striking contrast between these curves. True, there is a gradual initial rise in the curve for malaria-treated patients, but after three years the curve is entirely flat. One year after onset of symptoms, only 9 percent of the malaria-treated group were blind as compared with 30 percent of the control group; after two years only 14 percent as contrasted with 50 percent; after three years only 18 percent as contrasted with 60 percent. Thereafter for observation periods up to 15 years no additional blindness ensued in the malaria-treated group, whereas at that time period after onset of symptoms 90 percent of the control group were blind.

It will be recalled that despite selection of material no valid therapeutic claims could be made for either routine or subdural therapy. Here, however, we have an

* Since these data were prepared, an additional group of patients has been treated with initial results over a period of one to two years conforming to the good results described herein.

apparently striking therapeutic result. It is therefore essential to examine in detail the question of selection of material, particularly with reference to the malaria-treated group. In the section on critique of material it has already been shown that the course of the untreated disease had to be estimated from anamnestic data. The data for the malaria group during the period of their disease following treatment is, on the other hand, based upon actual observation, with determination of visual acuity before treatment and at the end of the observation period. We must therefore conclude that the basic data for the two groups of patients are not strictly comparable.

It has been demonstrated (figs. 1 and 2), moreover, by a comparison of the rate of progress of visual failure prior to treatment that patients later given subdural or malaria therapy represent a selected group of slow progressors as compared with patients never treated or subsequently given routine treatment only. A more detailed consideration of the factors of selection in the malaria group follows.

Of the 241 patients in the control group as previously defined, 29 were subsequently given malaria therapy. Otherwise stated, 29 of the 32 malaria-treated patients are also included in the control group for that period of time between onset of symptoms and the institution of fever therapy. It is therefore possible to compare the course of these 29 patients *prior to* fever therapy with the course of the remainder of the control group. This is done in the scatter chart of figure 9. The observations recorded are those immediately prior to fever treatment for the malaria group, and the last-recorded observation for the untreated

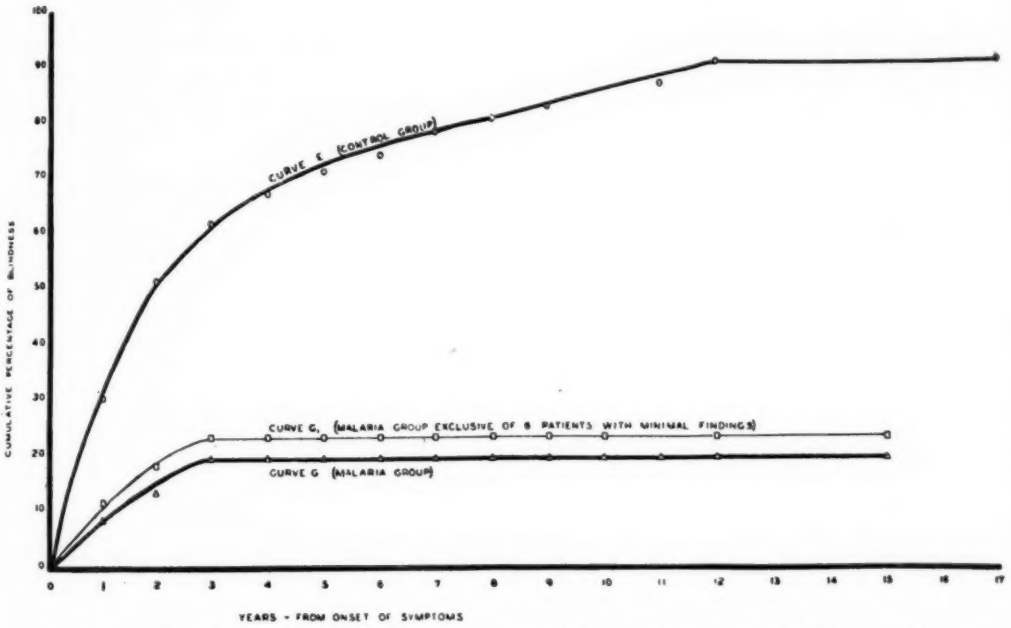
CURVE E (CONTROL GROUP)
CURVE G, (MALARIA GROUP EXCLUSIVE OF 6 PATIENTS WITH MINIMAL FINDINGS)
CURVE G (MALARIA GROUP)
CUMULATIVE PERCENTAGE OF BLINDNESS
YEARS - FROM ONSET OF SYMPTOMS

Fig. 8 (Moore *et al.*). Cumulative percentage of blindness in malaria group (curve G) compared with cumulative percentage of blindness in total control group (curve E). Elimination from the malaria group of six patients with minimal ophthalmologic findings does not materially alter the trend (curve G_1).

and routinely treated groups. Patients later given subdural treatment are included in the latter group, the recorded observation being the last before the institution of intraspinal therapy. For practical purposes figure 9 may be considered to represent the course of the untreated disease. A study of this scatter chart reveals two basic facts of major importance. First, the course of primary optic atrophy is extremely variable. The extremes of variability are illustrated by 33 patients who were blind less than 6 months after onset of symptoms and 14 patients who were not blind 5 or more years after onset of symptoms. Second, and of even greater importance, for any given time period after onset of symptoms the visual acuity was better maintained in patients subsequently given malaria than in the remainder of the control group.

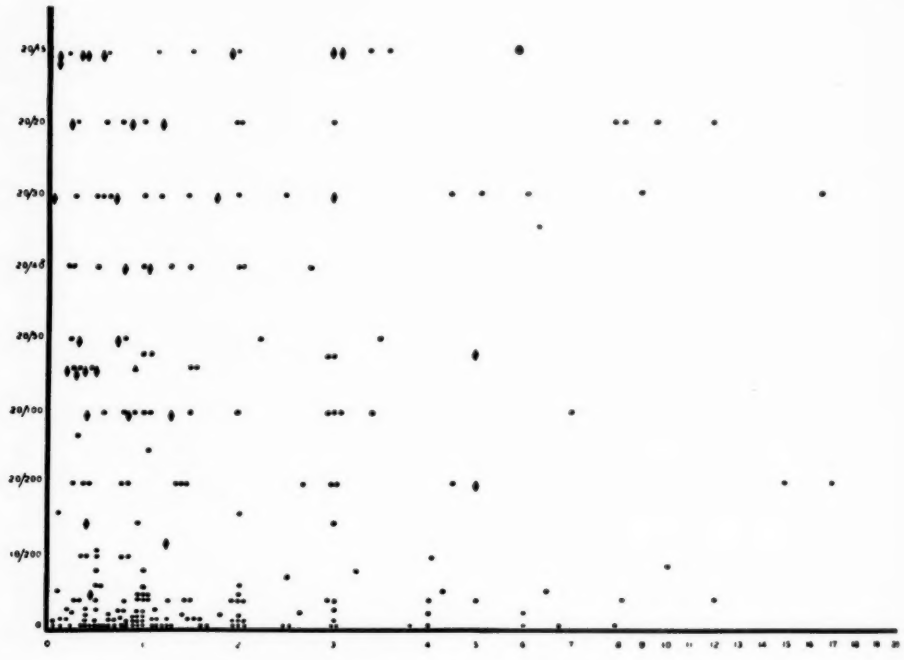

Fig. 9 (Moore *et al.*). Relationship between duration of symptoms and visual acuity in control* group. Scatter chart demonstrating that for any given time period after onset of symptoms visual acuity was better maintained in 29 patients subsequently given malaria than in 209 patients comprising remainder of control* group.

Each observation represents an individual patient. Abscissa indicates elapsed time between onset of symptoms and termination of control* period. Ordinate indicates visual acuity in better eye. Dots = observations at end of control period in remainder of control group. Diamonds = observations at start of malaria therapy in patient subsequently given malaria.

Another method of demonstrating that patients subsequently given malaria were a selected group of slow progressors is to compare by the life-table method the cumulative percentage of patients in both the above groups whose vision dropped below a fixed level with passage of time

* The control group is composed of all person-years under no treatment or routine treatment. It therefore includes patients subsequently given malaria or subdural therapy up to the time either of these special forms of treatment was instituted. For details see text.

after onset of symptoms. Arbitrarily, 20/70 vision in the better eye was selected. Utilizing this basic figure instead of 10/200 vision, the derivatives of the primary figures were calculated in the same manner as in the preceding life tables. The results are presented in columns A and B of table 17, and compared graphically by curves A and B of figure 10. Four months after onset of symptoms none of the patients subsequently given malaria had less than 20/70 vision while 9 percent of the remainder of the control group had dropped below this level. After eight months the percentages were 14 and 21, after one year 20 and 39, after two years 39 and 62, after three years 39 and 72, and after three years 70 and 94, respectively.

TABLE 17

CONTROL GROUP
CUMULATIVE PERCENTAGE OF PATIENTS WITH LESS THAN 20/70 VISION

Months after Onset of Symptoms	A 209 Patients Never Given Malaria	B 29 Patients Subsequently Given Malaria	C 41 Patients Subsequently Given Subdural Treatment
4	8.8	0	2.6
8	21.0	14.0	8.7
12	39.2	20.1	19.5
24	61.9	39.0	28.5
36	71.8	39.0	52.1
More than 36	93.6	69.5	65.7

The marked disparity between the rate of progression of the untreated disease in patients subsequently given malaria and in the remainder of the control group, the extreme variability of the untreated disease, and the fundamental differences in the derivation of basic data preclude any accurate comparison between the two

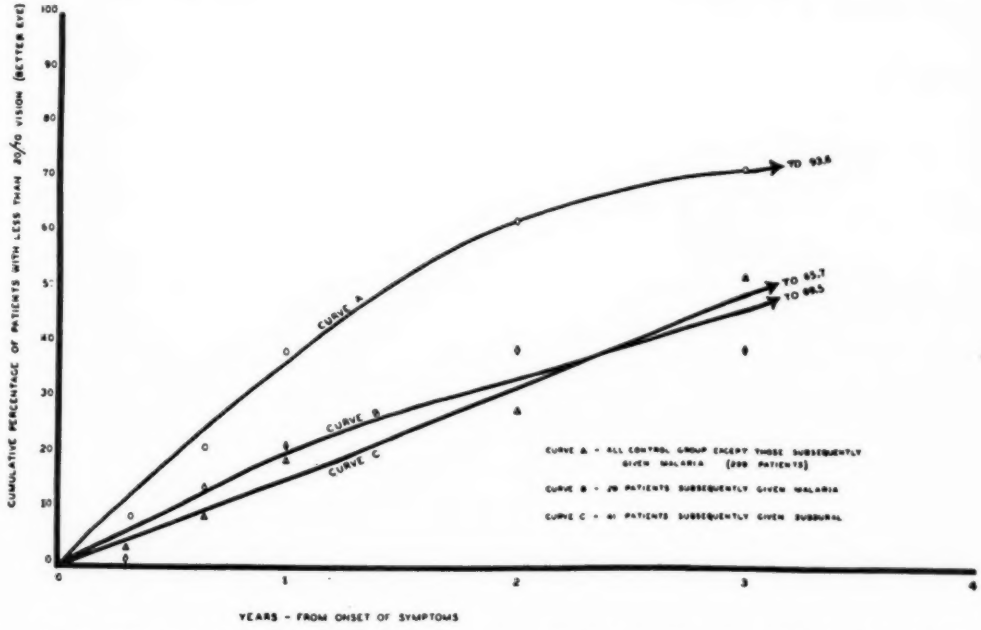

Fig. 10 (Moore *et al.*). Cumulative percentage of patients in control group with less than 20/70 vision. Demonstrating that the rate of progression of optic atrophy was less rapid in those patients subsequently given malaria than in the remainder of the control group. The rate of progression was essentially the same in patients subsequently given malaria as in patients subsequently given subdural therapy.

groups of patients. In effect, the control group is such in name only and not in fact.

Two alternatives are now open to us. We can discard all patients in the malaria-treated group except those with obviously rapidly progressing disease, or we can select from the control group a subgroup comparable to the malaria-treated patients. Since there were only 32 patients in the malaria group, in the majority of whom the disease was not rapidly progressive, the former alternative is not feasible. Fortunately the patients given subdural therapy form an admirable control group for those given malaria therapy. If it can be shown that these two series of patients are similar in all relevant respects except treatment, a significant difference in outcome must be attributable to difference in treatment. That the outcome is significantly different in the two groups has already been amply proved (compare fig. 6 and fig. 8). The essential similarity in other respects remains to be demonstrated. This is done in the scatter chart of figure 11 in which the observations recorded are those immediately *prior* to malaria and subdural therapy, respectively. A study of this scatter chart reveals that for any given time period after onset of symptoms the visual acuity was approximately equally maintained in the two groups of patients. This is even more clearly demonstrated by a comparison of curves B and C of figure 10 based upon columns B and C of table 17. Four months after onset of symptoms none of the patients subsequently given malaria and only 3

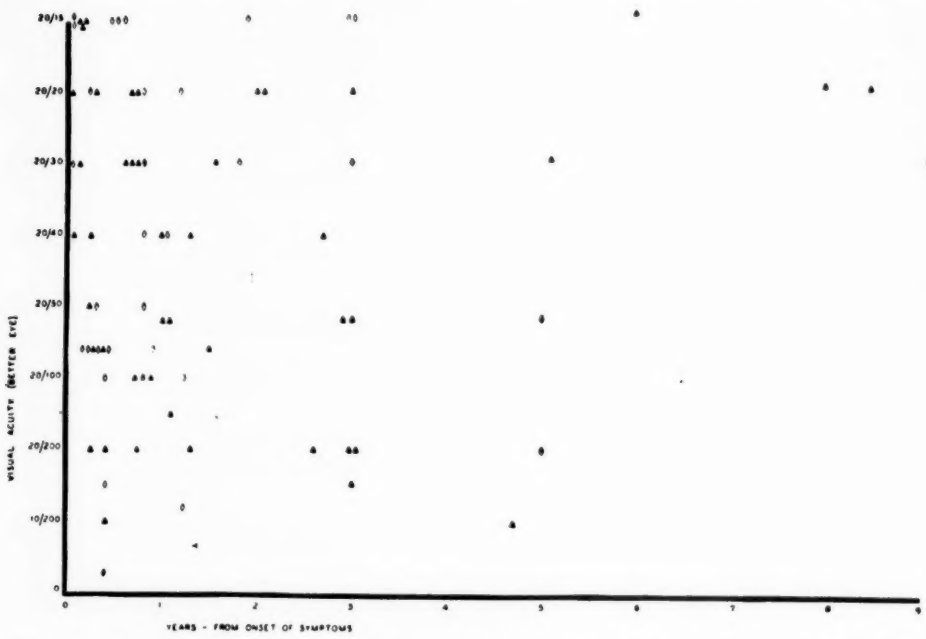

Fig. 11 (Moore *et al.*). Scatter chart comparing the course *prior* to treatment of 29 patients subsequently given malaria with the course *prior* to treatment of 41 patients subsequently given subdural therapy. For any given time period after onset of symptoms the visual acuity was approximately equally maintained in the two groups. Abscissa and ordinate are as in figure 9. ◇ = observation at start of malaria-therapy in patients subsequently given malaria. ▲ = observation at start of subdural therapy in patients subsequently given subdural therapy.

percent of those subsequently treated subdurally had less than 20/70 vision. After eight months the percentages were 14 and 9, after one year 20 and 19.5, after two years 39 and 28, after three years 39 and 52, and after three years 70 and 66, respectively. The curves are almost superimposable.

Each series contains approximately 10 percent of patients treated by subdural or fever therapy before onset of symptoms. There is, however, one discernible difference in that six patients had maintained normal visual acuity in both eyes prior to malaria treatment while there were only four similar patients in the subdural group. The diagnosis in these 10 patients was based upon slight to moderate changes in visual fields and more or less pallor of the optic discs noted in the course of routine ophthalmologic examination. The malaria and subdural groups of patients are similar also in that each contains approximately 10 percent of patients who had maintained vision for more than three years before special therapy was instituted. Here the selection is in favor of the subdural group which contains five patients who had maintained 20/30 or better vision for more than three years before the institution of subdural therapy while the malaria group contains only one similar patient.

It has thus been demonstrated (a) that the course of the disease prior to treatment was essentially the same in the malaria and subdural groups; (b) that both groups contain an approximately equal percentage of patients with very early optic atrophy; (c) that both groups contain an approximately equal percentage of patients with slowly progressive optic atrophy. The difference in outcome subsequent to treatment, based in both groups upon actual objective determinations, therefore is attributable to the difference in treatment itself, and the cumulative percentage of blindness as illustrated for the subdural group in curve F of figure 6 represents the best that can be expected in the absence of malaria therapy even in a selected group of slow progressors. Had these patients been given no treatment, the outcome might possibly have been even more unfavorable. At least 30 percent of this selected group would have been blind after one year, 50 percent after two years, 56 percent after three years, 61 percent after five years, and 75 percent 10 years after onset of symptoms. *Malaria therapy reduced the expected incidence of blindness in a similarly selected group to 9 percent after one year, 14 percent after two years, and 18 percent after three years with no further increase thereafter (curve G of fig. 8) for observation periods up to 15 years.* In other words, to the 25 percent of selected patients who would still have had vision 10 years after onset of symptoms, an additional 57 percent is added by fever therapy.*

Unilateral optic atrophy

In only 10 of the 250 patients under study could the diagnosis of unilateral optic atrophy be made at the time of initial examination, the normality of the opposite eye being based in each instance on normal disc color, visual acuity, and visual fields. Two of these patients were seen before onset of symptoms and with normal visual acuity in the affected eye as well. Six patients had had symptoms for less than eight months. Of these six, three were already blind in the affected eye. There were, however, two patients who

* By recalculation of experience exclusive of the six patients in whom the diagnosis was based only on early changes in visual fields and appearance of optic nerves, these percentages are not significantly altered (see curve G_1, fig. 8).

TABLE 18

PERCENTAGE DISTRIBUTION WITH RESPECT TO TYPE OF TREATMENT ADMINISTERED TO PATIENTS IN WHOM OPTIC ATROPHY WAS ASSOCIATED WITH TABES, PARESIS OR TABOPARESIS, AND MENINGOVASCULAR NEUROSYPHILIS

	105 Patients with Tabes (percent)	42 Patients with Paresis or Taboparesis (percent)	90 Patients with Meningovascular Neurosyphilis (percent)
No treatment	32	33	39
Routine only	37	21	31
Subdural ± Routine	20	20	14
Malaria ± Subdural ± Routine	11	26	16
Total	100	100	100

had had symptoms for 18 and 36 months, respectively.

One of these 10 patients remained untreated and was lost to further observation. Of five patients in whom routine therapy was the initial form of treatment, only one experienced involvement of the better eye. Under routine therapy alone, the disease in the other four patients remained unilateral for 10, 12, 24, and 72 months, respectively, after onset of symptoms. In view of the demonstrated inefficacy of this form of treatment this is probably an expression of the spontaneous course of the disease in these patients.

Malaria, subdural therapy, or both were given to eight patients at some time in the course of their disease. In one patient in whom the second eye had previously become involved, gradual progression culminated in blindness 16½ years after onset of symptoms. There remained, then, seven patients with unilateral disease at the time one of these special forms of treatment was instituted. After onset of symptoms, the disease remained unilateral in all for observation periods of 3, 5, 10, 12, 12, 14, and 16 years, respectively. In the first and third patients this cannot, however, be attributed to the treatment itself as one eye had remained normal for two and six years, respectively, before special treatment was instituted. In the latter four, at least, treatment would appear to be the responsible factor. In any small group of patients, however, cautious interpretation is necessary, in view of the extremely slow course the untreated disease may take in an occasional patient. This is well shown in figure 1 for five patients who maintained normal visual acuity for three or more years without treatment and in figure 9 for five patients who did as well for 6 to 12 years on routine treatment alone.

It is evident from the above discussion that except very early in the disease unilateral primary optic atrophy is unusual,

TABLE 19

CUMULATIVE PERCENTAGE OF BLINDNESS IN OPTIC ATROPHY ASSOCIATED WITH THE SEVERAL FORMS OF NEUROSYPHILIS

Years After Onset of Symptoms	105 Patients with Tabes	90 Patients with "Meningovascular" Neurosyphilis	42 Patients with Paresis or Taboparesis
1	26.8	29.7	29.6
2	46.6	45.9	40.9
3	51.6	54.1	50.2
4	55.9	55.6	56.8
5	57.4	59.2	60.4
6	60.7	61.2	68.0
7	60.7	65.5	68.0
8	64.6	67.8	68.0
9	66.9	70.1	68.0
10	66.9	72.5	68.0
11	66.9	75.0	68.0
12	66.9	77.8	68.0
13	66.9	77.8	68.0
14	66.9	77.8	68.0
15	66.9	77.8	68.0
16	66.9	77.8	68.0
17	66.9	82.7	
18	66.9	82.7	
19	66.9	82.7	
20	66.9	82.7	
21	66.9	82.7	
22	66.9	82.7	
23		82.7	
24		82.7	
25			

but that, in the rare case, the process may remain unilateral for as long as six years even in the absence of effective treatment. There is another small group of patients in whom, though the second eye may early become involved, progression is extremely slow. The necessity for large numbers of patients and long observation periods is thus reëmphasized.

consideration because, as shown in table 18, the percentage distribution with respect to type of treatment administered approximates equality. The only exception is that, for obvious reasons, malaria was given to a greater percentage of patients with paresis or taboparesis.

The cumulative percentage of blindness in optic atrophy associated with the sev-

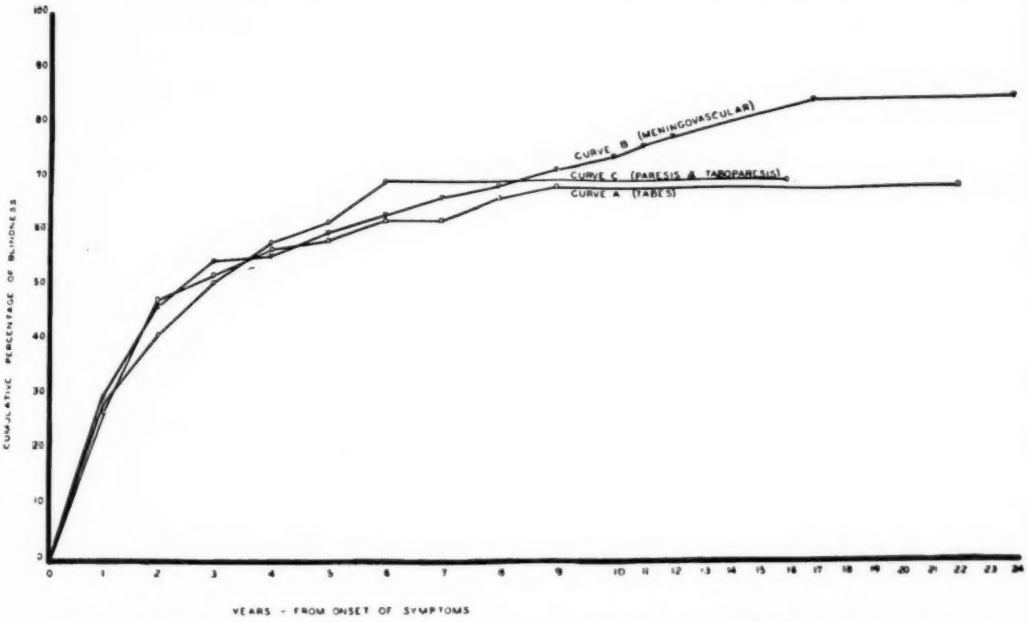
CURVE B (MENINGOVASCULAR)
CURVE C (PARESIS & TABOPARESIS)
CURVE A (TABES)
CUMULATIVE PERCENTAGE OF BLINDNESS
YEARS - FROM ONSET OF SYMPTOMS

Fig. 12 (Moore *et al.*). Cumulative percentage of blindness in optic atrophy associated with the several forms of neurosyphilis, demonstrating the similar prognosis in all groups. Curve A = 105 patients with tabes; B = 90 patients with meningovascular neurosyphilis; C = 42 patients with paresis or taboparesis.

The prognosis of optic atrophy associated with the several forms of neurosyphilis

Excluding the 13 patients in whom optic atrophy was associated with congenital neurosyphilis, there remain 105 patients with tabes, 42 patients with paresis or taboparesis, and 90 patients classified as meningovascular neurosyphilis because of the absence of posterior-column signs. In the comparison of these three groups of patients with respect to ultimate outcome, treatment can largely be eliminated from

eral forms of neurosyphilis is presented in table 19 and compared graphically by curves A, B, and C of figure 12. The essential similarity of these curves demonstrates that there is no significant difference in the prognosis of optic atrophy whether associated with tabes, paresis or taboparesis, or so-called meningovascular neurosyphilis. This is confirmatory of the opinion, earlier expressed, that there is no difference in the optic atrophy of tabes and that of so-called "basilar meningitis," certainly as to course when treated or un-

treated, and probably as to pathogenesis. The presence of optic atrophy alone is sufficient to indicate parenchymal involvement whether or not obvious posterior-column damage is present.

Prognosis of optic atrophy related to the type of visual-field defect

Sloan and Woods[11] have discussed fully the types of visual-field defects observed in patients with primary optic atrophy. These types and definitions of what we have termed "slight," "moderate," and "advanced" field defects are detailed in table 20. In tables 7, 8, 9, 10, and 11 (which provide the basic data for the several treatment groups) are shown, for the cases in which the information is available, the type and degree of visual-field defect at the start of, or early in, treatment, and at the final observation. In many patients the visual fields were carefully studied by one observer. Not-infrequent variations at the hands of different observers, failure to test for scotomata, omission of color-field determinations, and variation in the size of test object necessitated some latitude in classification. Although these factors, together with absence of data for many patients concerning visual fields before and after treatment, precluded the utilization of visual-field changes in the evaluation of treatment, a study of the basic data reveals, in general, that maintenance of visual acuity was accompanied by lack of change in the fields while progressive loss of visual acuity was paralleled by progressive changes in the fields. Thus, in 62 patients visual-field studies were made both before and after treatment. In 68 percent of these patients a change (or lack of change) in acuity was paralleled by change (or lack of change) in visual fields; in 16 percent the visual fields remained the same whereas visual acuity diminished, and in 16 percent the visual-field changes progressed whereas visual

TABLE 20

Definitions of Types and Stages of Field Defect—Slight, Moderate, and Advanced

Type I Concentric contraction with late involvement of central area. Sparing of central visual acuity until late stage	*Advanced*—Field for 1° red zero *Moderate*—Average limit for 1° white <30° Field for 1° red not reduced to zero *Slight*—Average limit for 1° white >30°
Type II Localized peripheral contraction. Central visual acuity may be involved somewhat earlier than in Type I	*Advanced*—Field for 1° red zero *Moderate*—More than half of field blind to 1° white Field for 1° red not reduced to zero *Slight*—Less than half of field blind to 1° white
Type III Central scotoma with normal peripheral fields. Central visual acuity reduced at onset	*Advanced*—Scotoma for 1° red has spread to periphery and field for this color zero. Caecocentral scotoma for 1° white *Moderate*—Caecocentral scotoma for 1° red, not for 1° white. 1° red seen in mid-periphery *Slight*—Caecocentral scotoma for 0.5° red, but not for 0.5° white
Type IV Central scotoma combined with peripheral defect of Types I or II	*Advanced*—Field for 1° red zero *Moderate*—As in Types I, II and III *Slight*—Caecocentral scotoma for 0.5° red, but not for 0.5° white. Slight peripheral defect as in Types I and II

acuity remained unchanged. In 8 of the 10 latter patients the progression was in the form of slight to moderate further changes in the peripheral fields. Our use of visual acuity as a standard measure of the efficacy of treatment thus seems justified.

The material presented in prior sections of this study has shown the extreme variability in the rate of progression of primary optic atrophy. This variation was found to be greater than could be accounted for by differences in treatment alone. At least one additional factor which determines the ultimate outcome is the type of field defect.

Of the 250 patients included in this study, 108 had visual fields taken at some time during observation. Many of the remainder, comprising the most rapid progressors, were already blind at the first observation. Sixty-nine of these 108 patients showed peripheral constriction of the visual fields without scotomata (types I and II) and the other 39 patients had central or paracentral scotomata (types III and IV). In the comparison of these two groups of patients with respect to ultimate outcome it is fortunately possible to eliminate treatment from consideration. This can be done because, although on a basis of type of treatment administered (table 21) patients with scotomata might be expected to make a slightly better showing, the converse was found actually to be the case.

TABLE 21

PERCENTAGE DISTRIBUTION WITH RESPECT TO TYPE OF TREATMENT ADMINISTERED TO 69 PATIENTS WITHOUT SCOTOMATA AND 39 PATIENTS WITH SCOTOMATA

	69 Patients without Scotomata percent	39 Patients with Scotomata percent
No treatment	17	13
Routine only	30	23
Subdural ± Routine	26.5	23
Malaria ± Subdural ± Routine	26.5	41
Total	100.0	100.0

The cumulative percentage of blindness for these two groups of patients is presented in table 22 and compared graphically by curves A and B of figure 13.

TABLE 22

CUMULATIVE PERCENTAGE OF BLINDNESS IN OPTIC ATROPHY RELATED TO THE TYPE OF VISUAL-FIELD DEFECT

Years After Onset of Symptoms	69 Patients without Scotomata percent	39 Patients with Scotomata percent
1	10.5	15.8
2	15.5	15.8
3	17.4	18.1
4	19.5	25.4
5	26.7	25.4
6	29.3	34.2
7	29.3	39.1
8	29.3	39.1
9	32.7	44.9
10	36.7	44.9
11	36.7	54.1
12	36.7	54.1
13	36.7	54.1
14	36.7	54.1
15	36.7	54.1
16	36.7	54.1
17	36.7	77.0
18	36.7	77.0
19	36.7	77.0
20	36.7	77.0
21	36.7	77.0
22	36.7	77.0
23		77.0
24		77.0
25		77.0

During the first five years the two curves are almost superimposable. Seven years after onset of symptoms 30 percent of patients without scotomata and 40 percent of patients with scotomata were blind. Thereafter there is a gradual divergence so that after 9 years the percentage blindness is 33 and 45, and after 11 years, 37 and 54, respectively. There was no further increase in blindness among patients without scotomata while, after 17 years, 77 percent of patients with scotomata were blind. It may thus be concluded that, in general, the prognosis in primary optic atrophy is less favorable

in the presence of central or paracentral scotomata. This is entirely in accord with what would be expected, since a central scotoma indicates involvement of the nerve fibers supplying the macula, and a similar outcome may result from slight further spread of a paracentral scotoma.

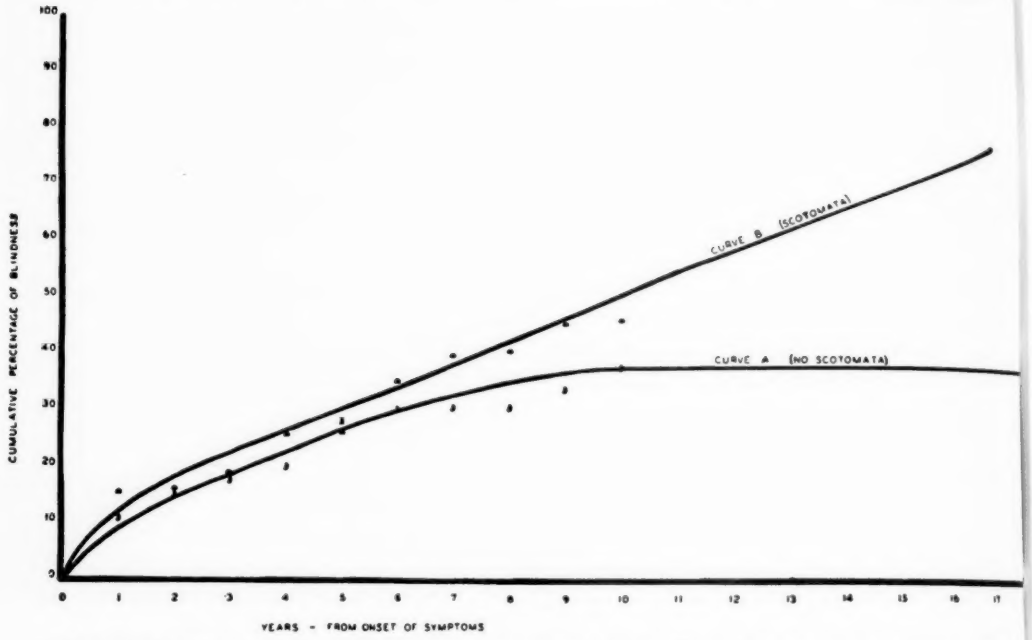

Fig. 13 (Moore *et al.*). Cumulative percentage of blindness in 69 patients without scotomata and in 39 patients with scotomata, demonstrating the less favorable prognosis in the latter group.

On the other hand, an equal degree of progression with respect to involvement of nerve fibers may be without practical significance when limited to the peripheral field.

The deleterious effects of treatment

The use of many millions of doses of the trivalent arsenical products the world over has never, in any authenticated instance, resulted in damage to a previously normal optic nerve. The same holds true for bismuth, mercury, and the iodides. In our experience, routine treatment alone does not alter the spontaneous course of optic atrophy for better or for worse. Patients maintained vision or went blind regardless of, and not because of, routine treatment. Subdural treatment, as previously pointed out, is a different case, carrying a risk of sudden progression of the atrophic process even to the point of blindness in about 10 percent of patients treated. This, coupled with the absence of statistically significant beneficial effect, renders the method undesirable. We have observed no sudden progression of the disease after malaria therapy. One patient (no. 37 of table 11) became blind one month after the institution of malaria, but this cannot be attributed to treatment because the vision in the better eye had fallen in 11 months to only 16/200 before fever therapy.

With few exceptions, we have not used tryparsamide in the treatment of primary optic atrophy because of the previously reported opinion that this drug is contraindicated in the presence of preëxisting damage to the nerve. Six patients were, however, given tryparsamide, alone in one instance, and as a supplement to malaria or subdural therapy in the re-

mainder. In only one of these (patient no. 9 of table 10) was there a definitely deleterious effect, consisting of rapid visual-field constriction. This did not, however, result in blindness.

Adhesive optochiasmal arachnoiditis in this material

The literature concerning the possible role of adhesive optochiasmal arachnoiditis in the etiology of syphilitic primary optic atrophy was reviewed in 1940 by Moore and Woods;[2] and more recently (1941) Hausman[12] has recorded further personal experiences. In view of the interest attaching to this syndrome, it is worth while recording that Dandy has performed exploratory operation on three of our patients in this series. In each of these, visual failure had been progressive in spite of fever and other forms of therapy and operation was undertaken as a last resort. In none was there any evidence of adhesive arachnoiditis, and the operative results were neither beneficial nor deleterious.

In spite of these negative results in three cases, we believe that in patients with syphilitic primary optic atrophy in whom visual failure is progressive in spite of induced malaria and subsequent chemotherapy, surgical exploration of the chiasmal area is justified. In the hands of a competent neurosurgeon, the operation has a low mortality; if adhesions about the chiasm are found their release may be of value and, in any case, the actual and now disputed relationship of the entity can be established.

Summary

1. Primary optic atrophy was found to occur in at least 8 percent of all patients with clinical evidence of acquired neurosyphilis. The incidence was higher in the neurosyphilitic Negro than in the neurosyphilitic white patient.

2. There appears to have been no significant decrease in the relative incidence of syphilitic primary optic atrophy during the past two decades.

3. The cerebrospinal fluid was found to be normal in nearly 7 percent of all cases.

4. A presumptive history of early syphilis was obtained in 59 percent of the male patients, and in only 8 percent of the female patients. The incubation period of the disease varied widely, though the majority of patients had onset of symptoms during the second decade of their disease. Only 1.3 percent of all patients had received adequate treatment for early syphilis.

5. Primary optic atrophy occurred more than twice as frequently in tabes as in other types of neurosyphilis. In uncomplicated paresis, it was found to be quite rare. Many patients, however, presented primary optic atrophy and pupillary changes as the only clinical manifestations of neurosyphilis. An analysis with respect to appearance time, rate of progression, and cerebrospinal-fluid findings failed to disclose any significant difference between this group and the group with clinical tabes.

6. The therapeutic results in 250 patients were subjected to statistical analysis by the life-table method. The course of untreated optic atrophy had of necessity to be estimated from anamnestic data influenced by a multiplicity of extraneous factors. Blindness occurred in 28 percent of untreated patients within one year of onset of symptoms, in 50 percent within two years, and in almost 65 percent within three years. Not until six years after onset of symptoms were 75 percent of patients blind, and less than 90 percent were blind at the end of 12 years. Patients not blind at the end of three years subsequently did quite well, even in the absence of treatment.

7. Routine antisyphilitic treatment with

the trivalent arsenicals and bismuth failed to alter significantly the spontaneous course of the untreated disease. The demonstrated inefficacy of routine treatment permitted the consideration of person-years under routine treatment as a unit with person-years under no treatment. Analysis of this total control group revealed extreme variability in the course of the disease even in the absence of effective treatment, as illustrated by 33 patients who were blind less than six months after onset of symptoms, and 14 patients who were not blind five or more years after onset of symptoms.

8. As estimated from the course of their disease prior to treatment, it was found that patients given subdural or malaria therapy were a selected group of slow progressors.

9. Subdural therapy precipitated blindness in almost 10 percent of patients. For the first 3 years after onset of symptoms the cumulative percentage of blindness was essentially the same in the subdural group as in the control group. Thereafter, there was a slight but widening difference, so that after 12 years 75 percent of patients given subdural therapy and 90 percent of the control group were blind. An analysis of the factors of selection appeared to vitiate this slight difference.

10. Only 9 percent of patients given malaria therapy were blind one year after onset of symptoms, only 14 percent were blind after two years, and only 18 percent were blind after three years. Thereafter, for observation periods up to 15 years, no additional blindness ensued. Selection of material and fundamental differences in the derivation of basic data, as well as the extreme variability in the course of the untreated disease, precluded accurate comparison between these patients and the control group. The demonstration that the course of the disease prior to treatment was essentially the same in the malaria and subdural groups, and that both groups contained an approximately equal percentage of patients with early disease and with slowly progressive disease, permitted the utilization of the subdural group as a control for the malaria group. It was demonstrated that to the 25 percent of these selected patients who would still have had more than 10/200 vision 10 years after onset of symptoms, an additional 57 percent was added by fever therapy.

11. In only 4 percent of all patients was the optic atrophy unilateral at the time of initial examination. It was difficult to assess the value of treatment in this small group.

12. The variation in the course of optic atrophy was found to be greater than could be accounted for by differences in treatment alone. At least one additional factor that determined the ultimate outcome was the type of field defect. In general, the prognosis was found to be less favorable in the presence of central or paracentral scotomata.

13. In view of the interest in the possible significance of adhesive arachnoiditis in the pathogenesis of syphilitic primary optic atrophy,[2, 12] it should be noted that intracranial exploration was performed by Dr. Walter Dandy on three of our patients. In none of these was adhesive optochiasmal arachnoiditis found at operation, and in none was there either beneficial or deleterious effect from the operation.

Conclusions

1. Syphilitic primary optic atrophy occurs most frequently in association with tabes dorsalis, although together with pupillary changes, it not infrequently may constitute the only clinical evidence of neurosyphilis. It is probable that the fundamental process is the same in all types of neurosyphilis.

2. The course of the untreated disease is extremely variable. The process almost

always becomes bilateral soon after onset of symptoms, and 90 percent of untreated patients are blind within 12 years. There is a small group of patients in whom progression is quite slow.

3. Our data suggest that the development of syphilitic primary optic atrophy can be prevented by the adequate routine treatment of early syphilis.

4. Routine therapy with the trivalent arsenicals and bismuth is of no value in the treatment of syphilitic primary optic atrophy.

5. Subdural treatment by the Swift-Ellis technique should be abandoned. Its dubious value in an occasional patient is outweighed by a risk of sudden total loss of vision in about 10 percent of those treated.

6. Fever therapy (in our hands malaria, since we have had no experience with the use of mechanically induced fever in this connection) is the only efficacious method of treatment. If visual failure progresses in spite of fever therapy, no other form of therapy is likely to be of value. It must be emphasized that these conclusions are subject to specific limitations imposed by the material upon which this study is based. Since malaria therapy was, by and large, not given to patients with the most rapidly progressive type of disease, no statement is permissible as to its efficacy in this group. It is certain that many of our untreated and routinely treated patients would not have been benefited by any treatment; indeed in many there was literally no time for treatment. The fact remains that where there is time for treatment, malaria is of definite practical value. Whereas early diagnosis is often possible only by routine determination of the visual fields, fundi, and visual acuity, this procedure is recommended as a routine supplement to the clinical and laboratory examinations in all patients with neurosyphilis. Only by early diagnosis and the immediate induction of malaria is there any hope of arresting the most fulminating cases.

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SURGICAL TREATMENT OF VASCULAR DISEASES ALTERING THE FUNCTION OF THE EYES*

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The major portion of this discussion will deal with arteriovenous fistulas situated between the internal carotid artery and the cavernous sinus, and with anomalies of the internal carotid artery which were responsible for hemianopia and loss of visual acuity. In presenting this subject I wish to emphasize certain personal observations and to discuss surgical procedures employed and results obtained rather than to give the usual review of each lesion that could be included under this title.

Pulsating exophthalmos

The medical literature dealing with pulsating exophthalmos is extensive, and a manuscript in which credit is given to each contributor would become too voluminous. However, it should be remembered that Benjamin Travers is credited with having performed in 1809 the first ligation of the internal carotid artery in the neck for the purpose of eliminating the arterial blood flow through the fistulous opening into the cavernous sinus, in the hope of relieving the symptoms of pulsating exophthalmos. Dandy,[5] in 1935, made an additional contribution by attempting to trap the fistulous opening by ligating the internal carotid artery in the neck; he followed this procedure with craniotomy, which permitted him to obliterate the internal carotid artery on the cerebral side as the artery emerged from the cavernous sinus before it gave off the cerebral arteries. It was reported that Zeller performed a similar operation in 1911, but that the patient died. Hamby and Gardner[13] reported a case of this type in 1933.

A portion of this paper deals with the same procedure but with the addition of occlusion of the ophthalmic veins in the orbit in order to relieve patients who failed to secure complete relief by double ligation (trapping of the arteriovenous aneurysm of the internal carotid).

In their monograph de Schweinitz and Holloway[29] gave a critical review of cases of pulsating exophthalmos, reports of which they had collected. They dealt with the etiology, pathology,[6] symptoms, treatment,[9] results, and indications for surgical intervention, and these represent the surgical indications that are employed today. Sattler,[27] in 1920, and Wheeler[32] also collected cases and made excellent analyses of their groups. Matas[21] emphasized the importance of gradual compression of the common carotid artery before attempting surgical procedure. He advised that the periods of compression be increased until compression could be maintained 30 minutes 3 times a day without producing syncope or hemiparesis. When this result was accomplished, reasonable assurance was established that adequate collateral circulation had developed. Locke,[19] Keegan,[18] and others devised appliances for compressing the carotid arteries. Partial constriction[25] of the common or internal carotid arteries with bands of metal or fascia has been resorted to when the patients have been unable to endure the temporary occlusion of the artery for 30 minutes. Brooks[2] suggested an ingenious method of introducing strips

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of muscle into the internal carotid artery, prior to ligation, with the hope of occluding the fistulous opening and stimulating thrombosis. This[18, 19] method has been only partially successful. Several contributions have been presented concerning techniques of ligation of the carotid arteries.[26, 28]

Once it has been established that it is reasonably safe to ligate the internal carotid artery, I prefer to ligate independently the common internal and external carotid arteries with fishline and to resect the intervening portion of the carotid arteries. The external carotid artery is ligated, inasmuch as it is known that the greater the flow of blood through it the greater is the possibility of establishing a reverse flow through the ophthalmic artery and other small branches of the internal carotid artery as it traverses the cavernous sinus.[30] In reports of attempts to control pulsating exophthalmos by ligating dilated pulsating ophthalmic veins without ligating the carotid arteries,[12] it was stated that bleeding was profuse and the attempt had to be abandoned. The

Fig. 1 (Adson). *a*, pulsating exophthalmos with proptosis and chemosis which resulted from an arteriovenous fistula of traumatic origin situated between the internal carotid artery and the cavernous sinus; *b*, same patient nine months after the ligation of the common carotid, external and internal carotid arteries in the cervical region.

ophthalmic veins are likely to remain dilated though somewhat reduced in size after the double ligation of the carotid arteries. Reid[25] opened the ophthalmic vein and applied 50-percent solution of glucose as a sclerosing agent. Singleton[30] injected solution of sodium morrhuate without incident.

The ligation of the internal carotid artery in the neck may be sufficient to relieve the symptoms of pulsating exophthalmos (fig. 1*a* and *b*). This is true especially if the proptosis is mild, a state which indicates that the fistulous opening is small. In the traumatic group,

Fig. 2 (Adson). Recurrence of symptoms of pulsating exophthalmos following trapping of an arteriovenous aneurysm situated between the internal carotid artery and the cavernous sinus. Photograph was taken five years after the primary operations.

however, which represents about 75 percent of cases of this type, cervical ligation of the internal carotid artery is not usually adequate although it may give some temporary relief.

The double ligation, trapping the arteriovenous aneurysm by the additional intracranial ligation of the internal carotid artery as it emerges from the region of the cavernous sinus before the major branches are given off, reduces the incidence of failures. There occasionally still

persist mild bruits, engorged ophthalmic veins and proptosis with frequent ophthalmoplegias and visual impairment. The photograph of the patient in case 1 illustrates such a result (fig. 2).

Obliteration of ophthalmic artery after the trapping of aneurysms

In these cases the persistance of symptoms prompted me, at the risk of destroying vision, to obliterate the ophthalmic artery by applying silver clips to the artery just as it arose from the internal carotid artery. This was done after intracranial obliteration of the internal carotid but at the same operation, for it was observed that a bright-red dilated ophthalmic artery pumped arterial blood into the ligated segment of the internal carotid. Although the volume of blood from the ophthalmic artery was small in comparison to the volume carried by the internal carotid artery, it was sufficient to maintain an engorgement of the ophthalmic veins. Obliteration of the ophthalmic artery immediately resulted in cessation of the remaining bruit and disappearance of pulsations in orbital structures. The tremendously dilated ophthalmic veins had subsided in size, but it was obvious that in order to obtain a cosmetic result I must resort to some means of obliterating or removing the enlarged ophthalmic veins—by intravenous injection of a sclerosing agent, by multiple ligations, or by venesectomy.

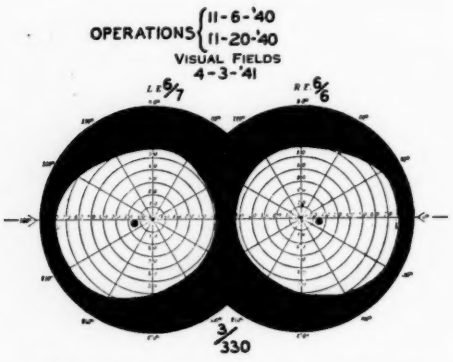

Fig. 3 (Adson). Perimetric fields made following trapping of an arteriovenous aneurysm situated between the internal carotid and the cavernous sinus, with intracranial obliteration of the ophthalmic artery and venesectomy of the ophthalmic veins in the orbit.

The superior and inferior ophthalmic and angular veins were exposed through three small incisions. The veins in turn were liberated, withdrawn, ligated, and resected, much as they are when varicose veins are stripped from a patient's legs. Care was taken to avoid injury to other orbital structures. At the conclusion of the operations, the proptosis had disappeared and all dilated veins had been removed.

When the patient regained consciousness, vision on the side of the pulsating exophthalmos was not impaired by the operation (fig. 3). The surgical procedures had consisted of cervical ligation of carotid arteries, intracranial obliteration of the internal carotid artery with silver clips, obliteration of the ophthalmic artery, and venesectomy of the ophthalmic veins.

In each of a series of four cases, three of which are not reported here, I ligated the internal, external, and common carotid arteries in the cervical region prior to ligation, intracranially, of the internal carotid artery on the cerebral side of an arteriovenous fistula. In addition, in cases 2, 3, and 4, the ophthalmic artery was occluded with silver clips and a venesectomy of the ophthalmic and periorbital veins was performed.

Case 2. The patient, a white man, married, twenty-five years of age, registered at the clinic January 24, 1940, complaining chiefly of pulsating exophthalmos on the left side.

On November 5, 1934, the patient had been in an automobile accident in which he received a head injury. That resulted in a fracture of the skull involving the base. There were hemor-

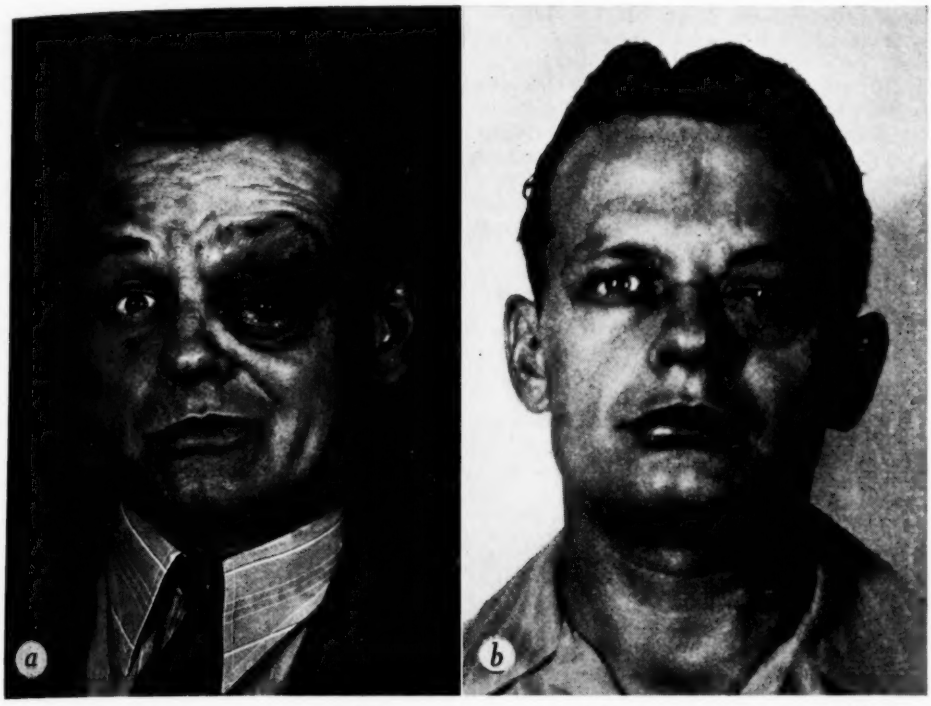

Fig. 4 (Adson). *a*, pulsating exophthalmos of traumatic origin due to a fistula situated between the internal carotid artery and the cavernous sinus on the left side; symptoms returned following cervical ligation of the internal and external carotid arteries. *b*, same patient after trapping of the arteriovenous aneurysm, but in addition the ophthalmic artery was obliterated intracranially at the same time that the internal carotid artery was ligated on the cerebral side of the arteriovenous fistula. Venesectomy also was performed in conjunction with the obliteration of the carotid and ophthalmic arteries. Vision was preserved.

rhages from the nose, mouth, and ears; the patient was unconscious for three days and continued to bleed from the ears until the twelfth day. On the sixteenth post-traumatic day he heard a swishing sound which on auscultation could not be detected as a bruit. Thirty-five days after the accident there had developed suddenly a protrusion of the left eye with palsy of the external rectus muscle. The bruit now became audible, and a diagnosis of an arteriovenous fistula was made. The opening of the fistula was discovered between the internal carotid artery and the cavernous sinus on the left side.

On December 15, 1934, ligation of the common carotid artery was effected by the patient's physician and surgeon, employing two ligatures of fascia for arterial constriction. The external carotid artery was ligated with silk. The patient was dismissed on the tenth postoperative day with marked reduction of the bruit. The veins about the eye and the soft tissues of the orbit extending over the orbital margin continued to pulsate.

Although after six months the symptoms of pulsating exophthalmos had not disappeared completely, the patient was able to return to work, and he continued to work until December 4, 1939, when he met with another injury that resulted in a fracture of the base of the skull with bleeding from the left ear and a marked increase in the exophthalmos of the left eye. He again was hospitalized for 11 days.

On January 24, 1940, he was admitted to the department of ophthalmology of the Mayo Clinic. Examination of the eyes revealed pulsating exophthalmos on the left side with marked proptosis and engorgement of veins within the orbit as well as engorgement of veins over the forehead in the temporal region and along the nose; the conjunctiva was congested and the lids were swollen. The left eye was displaced downward and outward (fig. 4*a* and *b*); the exophthalmometer reading was 34 in the left eye and 18 in the right eye. The vision of the right eye was 6/6 and the left eye 6/20. Examination revealed that the visual field of each eye was normal. There was no papilledema.

Roentgenologic examination of the head revealed an old fracture of the skull that extended from the posterior inferior region of the left parietal bone downward across the temporal bone into the region of the labyrinth and the petrous portion. The left orbit was slightly larger than the right with evidence of some thinning of its roof.

The results of laboratory studies were negative, as were those of the physical examination, with the exception of a right inguinal hernia, deafness in the left ear, pulsating exophthalmos of the left eye, and diplopia. Except for the indications just stated, the neurologic examination revealed normal conditions.

Attempts were made to obliterate the bruit by compression of the vessels in the neck, even though a previous ligation of the carotid arteries had been made. By compressing the jugular vein the bruit within the cranium could be altered.

Studies of the blood taken from the internal jugular vein revealed a concentration of oxygen greater than that ordinarily present in venous blood. Electro-encephalographic studies were made which disclosed normal conditions, suggesting that the collateral circulation was adequate. With the same apparatus, sound waves of the bruit were photographed.

The history of injury, the partial result from ligation of the carotid arteries in the neck, with an increase of symptoms following the second head injury, and the observations made at the Clinic compelled the conclusion that the pulsating exophthalmos was the result of a retrograde flow of arterial blood from the cerebral side into the arteriovenous fistula. It was apparent that if the patient was to be relieved of symptoms, this flow must be interrupted. In order to make sure that collateral circulation had not been established between the vessels in the neck, a reëxploration of the carotid arteries was made on February 5, 1940. It was discovered that the vessels had been ligated and that collateral circulation had not developed. Inasmuch as collateral circulation had developed prior to the ligation of the carotid arteries in the neck, it was reasonable to

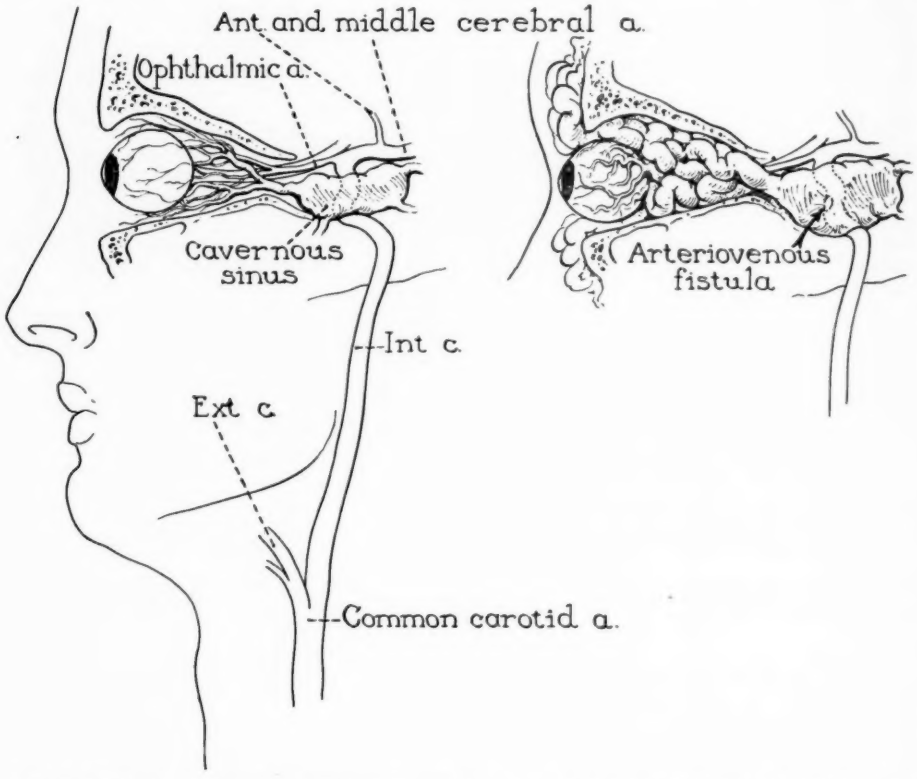

Fig. 5 (Adson). Schematic drawing illustrating the relationship between the internal carotid artery and the cavernous sinus and the proptosis and engorgement of the ophthalmic veins as a result of an arteriovenous fistula.

assume that intracranial ligation of the internal carotid was not contraindicated. I hoped to accomplish this ligation by a transtemporal approach, and on February 16th performed a decompression in the temporal region on the left side. I was able to elevate the temporal lobe and to see the bulging mass in the cavernous-sinus region situated extradurally, but I was unable to visualize the carotid artery on the mesial side of the mass. Therefore, I was compelled to close the wound and to advise the transfrontal approach which previously had been employed by Dr. Dandy,[7] others, and me.

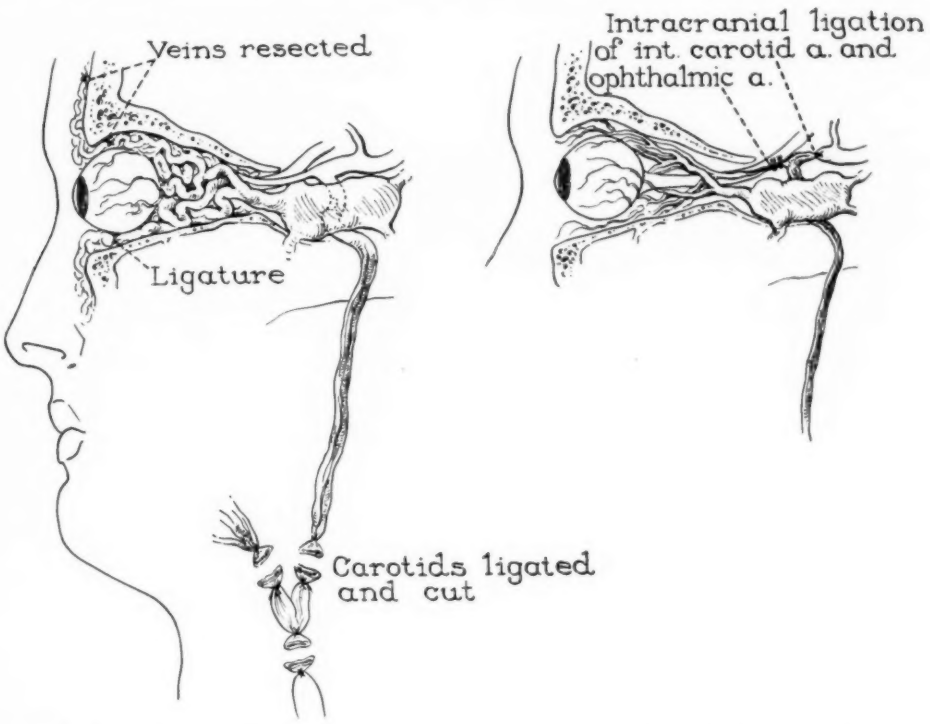

Fig. 6 (Adson). Schematic drawing illustrating the surgical steps in ligating the carotid arteries in the cervical region. Application of a ligature on the internal carotid on the cerebral side of the arteriovenous fistula, obliteration of the ophthalmic artery with silver clips, and venesectomy of the ophthalmic veins.

On February 21, 1940, a transfrontal, extradural approach was made for craniotomy. The dura was incised along the wing of the sphenoid in the anterior crest of the sella turcica, and I was then able to visualize the internal carotid artery as it emerged from the cavernous sinus through the dura as well as the arterial branches that arose from it (fig. 5). I was able to introduce a ligature (figs. 5 and 6) around the artery at this point and to effect an accurate tie with complete obliteration of blood flow in retrograde motion from the cerebral arteries. Subsequent to this I could observe arterial blood being pumped reversely from the ophthalmic artery into the trapped loop of the internal carotid artery. Having previously told the patient that vision might be impaired as a result of the intracranial ligation of the internal carotid artery and having obtained his permission to proceed with ligations of blood vessels as judgment warranted with the risk of visual impairment, I applied three silver clips to the ophthalmic artery, occluding with it a small arterial branch that apparently was directed from the carotid artery toward the hypophyseal region. The cranial wound was closed. On auscultation of the head, bruit could not be heard, and examination of the eye revealed that the proptosis had receded but that the dilated veins still were filled with blood and presented an unsightly appearance. It then occurred to me that it might be wise to perform a venesectomy (fig. 7), rather than to wait for spontaneous thromboses or to attempt the injection of the sclerosing agent that might do more harm than good.

Two small incisions were made, one over the

angular vein, the other over the superior ophthalmic vein. Loops of veins were liberated, withdrawn and resected until I was able to remove long portions from within the orbit from off the forehead, temporal, and nasal regions. In order further to stimulate localized thrombosis, electrocoagulation was applied to the cut ends of the veins. On completion of the venesectomy no venous masses remained in the orbit. The proptosis had disappeared. The wounds were closed with interrupted silk sutures. The eyelids were covered with gauze saturated with petrolatum and a dressing pad held in place by a roller bandage. This dressing was removed in 48 hours. The proptosis had disappeared; vision in the left eye had been preserved.

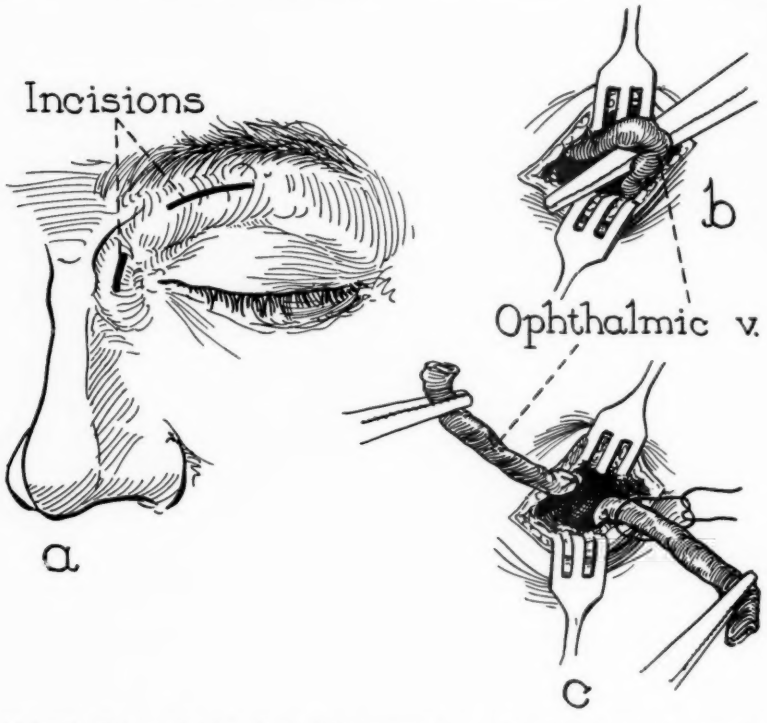

Fig. 7 (Adson). Schematic drawing of the surgical procedure employed in venesectomy of the ophthalmic veins; a, b, and c, steps in the operation.

A careful examination was made by an ophthalmologist on February 27th, six days after the operation. He reported that vision had not been disturbed, although there still was some chemosis and a papilledema of 1 diopter. Convalescence continued uneventfully, and the patient was dismissed from the Clinic on March 4, 1940.

The patient's physician at home reported on May 24, 1940, that the convalescence had continued uneventfully. Slight conjunctival injection and fullness under the eye remained, the bruit had not returned, and the proptosis had decreased fully three fourths of its extent at the time of the patient's admission. He stated also that binocular vision had returned and that the patient volunteered the information that he could read newspaper print with his left eye without difficulty.

On December 6, 1940, I had the privilege of seeing this patient, and I learned that he had returned to a gainful occupation. He was free from symptoms of pulsating exophthalmos, and although slight proptosis still persisted his vision was not impaired. He no longer was an object of curiosity and he had been accepted by his employer without fear of the employer's being responsible for any medical liability.

In the light of subsequent experience, I should have resected a loop of redundant veins from the inferior ophthalmic vein. In the future I shall not ligate the internal jugular vein in the neck as I did in this case, for if left open the veins and sinuses have a better chance to drain. The reason for ligating the internal jugular vein while exploring the carotid arteries was the fact that the bruit preoperatively had been reduced by manual compression.

Comment

To relieve the symptoms of pulsating exophthalmos,[14] whether unilateral or bilateral, it is obvious that a careful examination and an accurate diagnosis should be made. Moniz[23] and Terry[31] employed intravenous thorotrasts as diagnostic media. They, Dott,[10] and others[6] demonstrated roentgenographically aneurysmal lesions of the cerebral vessels. The procedure is reasonably safe and should be resorted to when it is impossible to differentiate the lesion by clinical means.[11, 15, 16, 17, 18, 23, 28, 29] If the lesion is caused by a fistulous opening between the intracranial portion of the carotid, or between a carotid aneurysm and the cavernous sinus, the treatment required is to reduce or obstruct the arterial flow to such an extent that all symptoms disappear. Ligation of the internal and external carotid arteries may be sufficient; if not, intracranial occlusion of the carotid arteries should be made. While intracranial occlusion is being accomplished the ophthalmic artery also should be occluded with silver clips. In my experience with four cases of unilateral pulsating exophthalmos, in which the arteriovenous fistula was trapped by a double ligation of the internal carotid artery, recurrence took place in the first case, and cure resulted in the three subsequent cases. The operative procedures in the second, third and fourth cases differed from those used in the first in that the ophthalmic artery was occluded and the enlarged tortuous redundant ophthalmic and periorbital veins were resected. Visual loss did not occur in these three cases. In the first case vision gradually diminished with the recurrence of symptoms and proptosis. Although intracranial occlusion of the ophthalmic artery is supposed to result in blindness, in this case it did not. Singleton[30] called attention to the copious collateral circulation of the ophthalmic artery within the orbit, which must explain why the vision of this patient was preserved.

Before attempting any ligation of the carotid arteries, the surgeon should heed Matas's[21] and Reid's[25] dictum of evaluating the collateral circulation: Proceed with graduated compression by digital or mechanical means until it is possible to occlude the artery for 30 minutes without untoward symptoms. If this is impossible partial occlusion of the carotid arteries with metal or fascial bands might be tried.

In the second case of this series of four cases a fascial band had been previously applied. When the carotid arteries in the neck were reëxplored it was observed that the band had given rise to considerable scar tissue and to complete occlusion of the common carotid artery.

Partial rotation with traction on the optic nerve caused by a fusiform enlargement of the internal carotid artery

Observation of partial rotation with traction on the optic nerve caused by a fusiform enlargement of the internal carotid artery was made during a reexploration of the optic chiasm for what was preoperatively believed to have been a recurrence of a chromophobe adenoma of the pituitary gland.

Case 5. The patient, a man, aged 42 years, seven years previously had presented the usual symptoms of a pituitary tumor. The tumor had been enucleated by the transfrontal intracapsular method. The patient recovered without incident, with restoration of vision in the temporal field of the left eye (fig. 8). Inasmuch as prior to operation, vision in the right eye had been negligible, with marked atrophy of the optic nerve, recovery did not take place. The patient enjoyed good health and assumed heavy responsibilities as an executive until a few months prior to the reëxploration, when he noticed that vision in his left eye had begun to fail. Although the visual loss was again chiefly in the temporal field of the left eye, this time

it was accompanied with pain located over the right eye.

Seven years and nine months after the original operation for pituitary tumor reëxploration of the chiasmal region was performed. As at the previous operation, a right transfrontal extradural approach was employed. Where the dura along the wing of the sphenoid and the anterior crest of the sella turcica was opened, it became obvious that there was present, not a ligate the internal carotid artery. Therefore I had made no attempt to obliterate the internal carotid artery. The only thing left to do was to free the adhesions, lift the nerve off the artery, and drop it into the recess mesial to the artery. This was done without injury. The left optic nerve appeared quite normal in size, color, and consistency, whereas the right nerve, although intact, was pale and soft.

The patient had a stormy convalescence. The

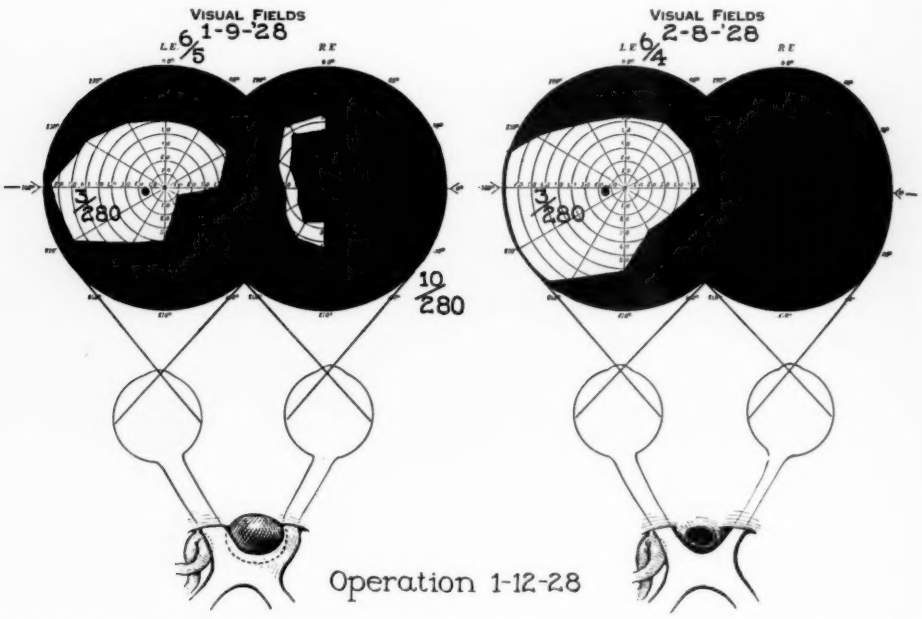

Fig. 8 (Adson). Case. 5. Perimetric fields before and after a transfrontal intracapsular enucleation of a chromophobe adenoma of the pituitary gland.

recurrence of the pituitary adenoma, but instead a vascular lesion. A fusiform enlargement of the left internal carotid artery had taken place, beginning as the artery emerged from the cavernous sinus through the dura. In the region of greatest enlargement the artery was about 1 cm. in diameter, and tapered down to nearly normal size at the point where the anterior cerebral artery arose (fig. 9). The left optic nerve was adherent to the artery. Thus it became apparent that the patient's symptoms were caused by adhesions, partial rotation of the left optic nerve, and traction on the nerve and chiasm.

The problem of treatment now confronted me. The patient had not complained at any time of bruit or murmurs, so that preoperative studies had not been made on carotid compression to determine the adequacy of collateral circulation in the event that it appeared advisable to

osteoplastic flap was elevated on the fourth postoperative day in order to rule out the presence of hemorrhage. None was found. Cerebral edema was distinct. The wound failed to heal and it was necessary to remove the bone flap. In spite of the eventful convalescence, vision in the temporal field in the left eye began to improve (fig. 9). Once the patient began to improve, he progressed satisfactorily. He fully recovered, regained most of his vision in the left eye, was relieved of pain over the right eye, and returned to his former position. At the time this paper was written, five years had elapsed since the second operation, during which time the patient had remained well and had assumed more responsibilities than he had carried prior to his operation. Yearly reëxamination had failed to show any untoward change in the vascular lesion or in the visual field.

Compression of Both Optic Nerves Due to Hardening and Internal Rotation of the Internal Carotid Arteries

During a craniotomy, while exploring the chiasmal region for an explanation of the presence of bitemporal hemianopia, I made an interesting observation of compression of both optic nerves due to hardening and internal rotation of the internal carotid arteries.

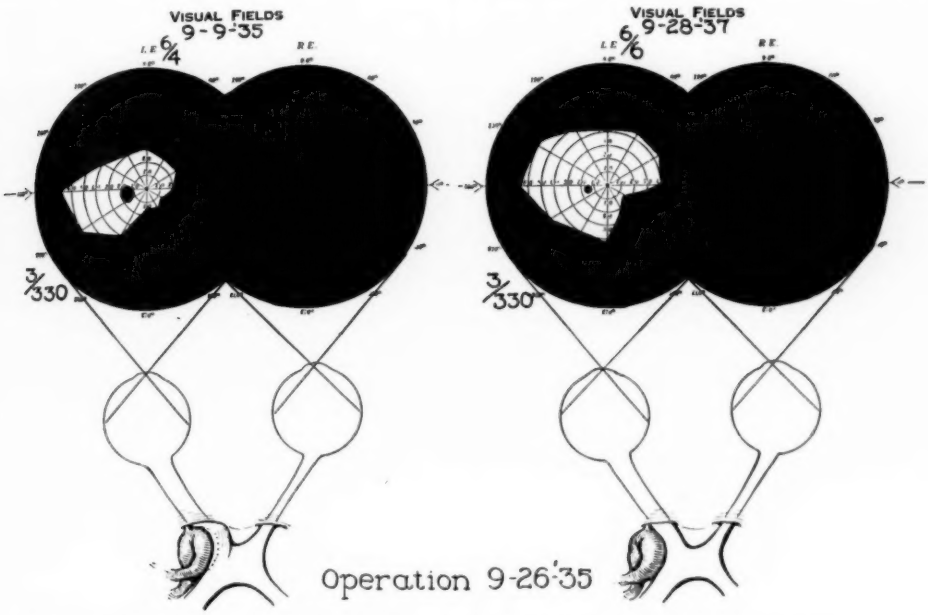

Fig. 9 (Adson). Case 5. Perimetric fields before and after reëxploration of the chiasma, with schematic drawing illustrating fusiform enlargement of the internal carotid arteries which was responsible for the field defect in the left eye and the relief obtained by liberating the left optic nerve.

Case 6. The patient was a woman, aged 49 years, whose chief complaint was that of failing vision. Until four months prior to admission she had enjoyed good health, when, although she had worn glasses for many years, her vision suddenly and rapidly began to fail.

When the ophthalmologist who examined the visual fields discovered that the patient had bitemporal hemianopia, he suspected the presence of a chiasmal lesion and referred her for surgical consultation. Examination in the ophthalmologic department verified the previous observations. Roentgenologic examination revealed that the sella turcica was normal. Symptoms of pituitary endocrine disturbance were not present. The results of general and neurologic examinations were likewise negative.

In view of the progressive nature of the patient's symptoms a craniotomy with exploration of the chiasmal region was performed through a right transfrontal extradural approach. The dura was opened along the wing of the sphenoid and the anterior crest of the sella turcica. There was evidence of neither a meningioma nor a pituitary tumor. Both optic nerves, however, were elevated and displaced mesially and upward in such a manner that the mesial border and the superior surface of both nerves were pressed against the inner and upper margins of the optic foramina. On elevating the optic nerves it became apparent that I had encountered an anomalous development of both internal carotid arteries (fig. 10). The arteries were large but not aneurysmal, they appeared to be thickened and hardened, and they varied from the normal in that each made a large loop upward and mesially before turning posteriorly. Because the walls of the vessels were thickened and stiffened, the arteries had the effect of foreign bodies under the optic nerves. This anomalous arterial change undoubtedly was responsible for the impingement of both optic nerves.

These anatomic findings presented an additional surgical problem. I tried to liberate the

optic nerves and place them mesially to the internal carotid arteries but found this impossible since the anterior portion of each arterial loop was located too far forward and mesially to permit sliding the optic nerves to the mesial side. My only hope of liberating the nerves was to unroof the optic canals in order to relieve the constriction or rather the indentation of the optic nerves on their mesial and upper surfaces. As the unroofing procedure was carried into each orbit it became apparent that I could achieve my purpose. Sufficient bone was removed to mobilize thoroughly each optic nerve. To assure further relief from constriction, the dural and arachnoid membranes were incised in the direction of the optic nerves. This procedure revealed the notch or indentation which had resulted from pressure against the mesial bony margins of each optic canal.

The postoperative results were most gratifying. The temporal field in the right eye made a complete recovery. The temporal field in the left eye made an acceptable gain although not a complete recovery (fig. 10).

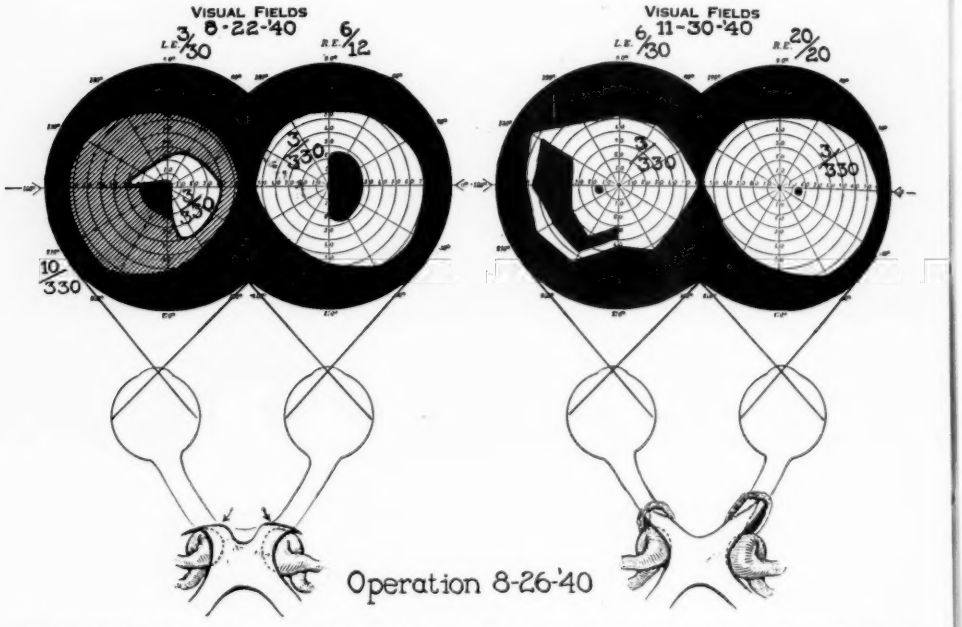

Fig. 10 (Adson). Case 6. Perimetric fields taken before and after operation. Schematic drawings illustrating the compression of the optic nerves against the superior mesial border of the optic foramen and the decompression of the optic nerves, with the resulting improvement in vision.

The surgical findings in another case were similar to those observed in the case just described and the operative procedures employed likewise were similar. The results were disappointing, owing to the fact that the visual loss had progressed until vision was negligible at the time of operation; after surgical intervention, slight gain developed in the right eye, but none occurred in the left eye. A brief review of the chronologic events in this case follows and illustrates how complicated the history, symptoms, and diagnosis can be. It may also emphasize the fact that if surgical intervention is to be instituted it should be resorted to before the patient's vision is lost.

Case 7. The patient, a married man, aged 31 years, was admitted to the Clinic on July 2, 1940. He gave a history of furunculosis in his youth; he had undergone appendectomy, and in 1934 left nephrectomy had been performed for stones.

He came to the Clinic chiefly because of blurring and diminution of vision, which had begun two months previously, after an automobile accident in which the rear of the patient's car had been struck, jerking his head backward. He did not receive any other injury.

He had not been aware of any adverse effect until on the second morning after the accident he had awakened with a headache; later in the day he had become conscious of blurring of his vision to the extent that he was unable to read the telephone book.

The patient's ophthalmologist had diagnosed bilateral retrobulbar neuritis and the patient had been hospitalized for further observation. Examination of the cerebrospinal fluid and neurologic examination gave negative results. Typhoid vaccine, potassium iodide, calcium, and vitamin B were administered and dehydration was employed, all without result. The patient was referred to the ophthalmologic department of the Mayo Clinic where the diagnosis of bilateral retrobulbar neuritis was confirmed. A trial course of histamine was given at the patient's request, with some temporary improvement of the visual fields. A series of 53 injections then was given with some improvement in the peripheral fields. A period of rest from the regimen of histamine was given, and during this time the visual fields promptly regressed to their original status. In view of this result and the probability that histamine only reduced the reactive edema of a still active inflammatory process, coupled with the inability to rule out absolutely the presence of a surgical lesion, an exploratory operation was advised and at the insistent request of the patient was carried out on March 5, 1941.

Physical examination at the Clinic was not significant except for mild hypertension and old scars. The blood pressure measured 140 mm. Hg systolic and 90 diastolic. The pulse rate was 78 per minute. The temperature was 98.6°F. (37°C.).

The results of routine laboratory studies were negative. The concentration of urea was 14 mg. per 100 c.c. of blood. Urinalysis gave negative results for arsenic; tests of urine for lead revealed 0.14 mg. in a 1,200 c.c. specimen, but tests of subsequent specimens repeatedly gave negative results.

Examination of the eyes revealed that vision in the left eye was 14/141 and in the right, 14/89. Pallor in both discs was graded 1 nasally and 3 temporally, on a basis of 1 to 4 in which 1 indicates the lowest and 4 the highest degree; the only vision remaining was peripheral, more on the right.

The results of neurologic examination, other than for visual disturbance, were negative.

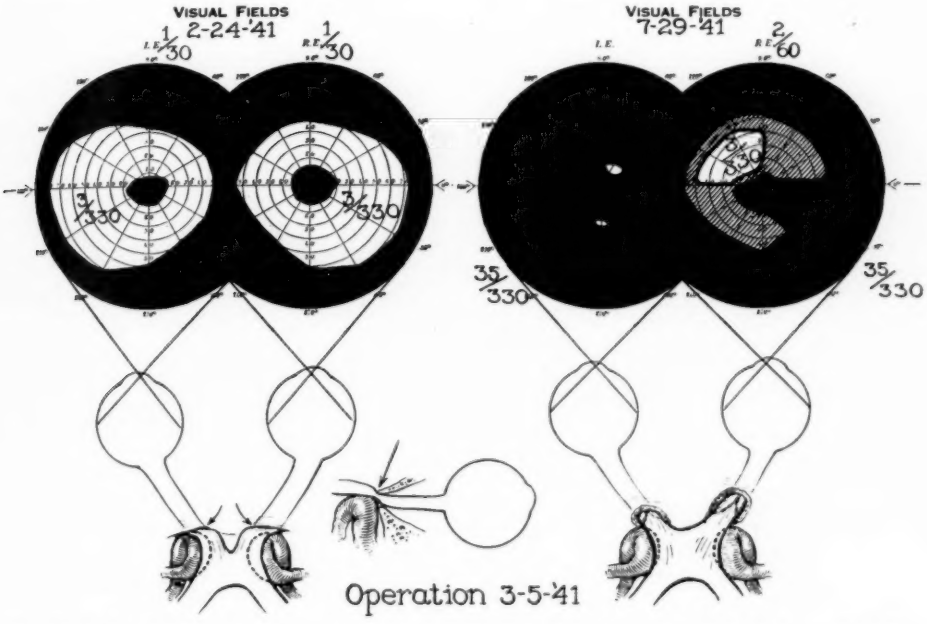

Fig. 11 (Adson). Case 7. Perimetric fields before and after an exploration of the optic chiasm. February 2, 1941, treatment with histamine improved vision temporarily but it failed progressively before operation. Schematic drawing illustrating the anomalous development of the internal carotid arteries, the decompression of the optic nerves performed and the continued loss of vision as a result of ischemia and fibrosis of the optic nerves from the trauma produced at the optic foramen.

Roentgenologic examination of the head revealed slight calcification of the falx, and two small regions of sclerosis in the left posterolateral portion of the frontal bone. The optic canal was normal.

A right transfrontal craniotomy was performed. Examination revealed that both optic nerves were angulated sharply by the internal carotid artery on either side. The arteries were situated ventral and lateral to the optic nerves, so that each nerve was compressed against the upper margin of the optic foramen. A small part of the left optic canal roof was removed and the entire roof of the right canal was taken away. The dural sheath of each optic nerve then was opened (fig. 11). Although the circulation to the nerves was impaired at the foramina, the remaining portion of the nerves and the chiasm appeared to have a moderate blood supply. Both the nerves were pale, but the right looked more nearly normal than the left. There were numerous adhesions between the nerves, chiasm, and arachnoid.

After surgical intervention, although the patient believed that he had received some improvement in vision, the results were so slight that he again was placed on medication. More histamine, acetylcholine, nicotinamide, and padutin were given. At that time (two and one-half months after operation) there was evidence of extremely slight improvement in the right visual field, whereas the fundi did not show any change. The patient was dismissed to return home on a regimen of nicotinic acid.

Aneurysms of the chiasmal region

Aneurysms occurring in the chiasmal region develop in the internal carotid, middle cerebral, anterior cerebral, anterior communicating, and, occasionally, in the ophthalmic artery. The lesion may be a fusiform enlargement of an artery, a sacculated mass, a pedunculated pouch, or it may be an arteriovenous aneurysm. Enlarged veins and sacculated deformities of veins also may produce symptoms.[20, 22]

The symptoms produced by an aneurysm vary according to the size and position of the aneurysm and to the artery involved. Small aneurysms may not produce any symptoms, others produce local symptoms of pressure with bruit, and the larger lesions usually produce contralateral paresis, increased intracranial pressure, bruit, and local signs of vascular disturbances and pressure.[16, 25] Roentgenologic studies with or without the use of thorostrast are of inestimable value in arriving at a differential diagnosis.

Lesions of the internal carotid artery have been controlled by ligation of the artery in the cervical region. Occasionally it is possible to trap the aneurysm by ligating the internal carotid artery intracranially as well as in the cervical region. Again it should be borne in mind that ligation of the internal carotid artery should not be undertaken without evaluating the collateral circulation. Dandy[8] reported being able to close the neck of a pedunculated aneurysm with silver clips. Dott[10] reported having trapped an aneurysm of the anterior communicating artery of the circle of Willis by ligating the artery on each side of the lesion.

No general rule can be made to apply for all aneurysms of cerebral vessels. Each case must be considered independently. The surgical indications are based on the severity of symptoms, the exact location and size of the lesion and the possibility of occluding the artery involved without producing paresis and a fatal termination.[3, 4]

Angiomatous tumors

Angiomatous tumors occur more frequently in the retrobulbar space than they do in the chiasmal region. Meningiomas and hemangio-endotheliomas[1] have a tendency to develop along the wing of the sphenoid, along the anterior crest of the sella turcica, and in the olfactory groove. Tumors of this group are extremely vascular. They frequently give rise to hyperostosis and invade the bone at the site of origin. It is not uncommon at surgical operation to find the roof of the orbit hypertrophied; when hypertrophy has occurred the change has extended in all directions. An osteoma not only will grow into the cranial cavity but also will fill the retrobulbar space. Thus,

a variety of symptoms may develop. Primary tumors in the retrobulbar space of the orbit are capable of producing proptosis, oculomotor palsy, hemianopia, papilledema, and loss of vision. The tumors situated intracranially in the chiasmal region can produce similar symptoms, with the exception of proptosis, unless the lesion has invaded the orbital plate and extended into the orbit, when proptosis also will be present.

The surgical management of vascular tumors situated within the orbit posterior to the globe or situated in the middle fossa and in the anterior fossa is not unlike that employed for less vascular encapsulated tumors. The chief differences in treatment, resection and ligation of communicating blood vessels, are determined by the degree of vascularity of the lesion.[24]

When patients suffering from vascular disease that affects ocular function come to the Clinic presenting problems that can be solved or lesions that can be treated, they are referred, according to type, to one of two departments. Cases of orbital tumor, whether or not the tumors belong to the vascular group, are considered in both the ophthalmologic department and in the neurosurgical department. When the tumors are accessible by a frontal approach or by Krönlein's approach an ophthalmologist does the operation. Cases in which the tumors are not accessible by such approaches are referred to the neurosurgeons. The one exception to this rule concerns those intracranial tumors that have invaded the orbital plate and have grown into the orbit. In such an instance it appears wiser to approach the tumor from above, through a craniotomy wound, by means of which it is possible first to remove the intracranial portion of the tumor before unroofing the orbit and removing the orbital extension. This method lessens the chances of infection and permits the surgeon to complete the operation in one stage. The differential diagnosis of this lesion is facilitated by carefully taken roentgenograms. Lateral views with the affected side nearest the plate will invariably reveal hyperostosis in the roof of the orbit.

In view of the numerous contributions that have been made to the medical literature on orbital tumors, I shall refrain from a general discussion but will emphasize a personal observation or two. Bleeding during craniotomy for the removal of a vascular tumor situated in the chiasmal and orbital regions had always been a troublesome problem until I ligated the external carotid artery prior to the craniotomy but during the same operative period. This ligation can always be done with safety and it reduces the arterial bleeding by four fifths. Many neurosurgeons perform decompression at the base of the osteoplastic flap at the conclusion of an operation for removal of tumor. For many years I have found it helpful to use a temporofrontal decompression opening when contemplating a transcranio-orbital procedure. It has proved advantageous to unroof the orbit after removal of a sphenoidal-ridge meningioma when proptosis has been present in the absence of any hyperostosis, inasmuch as intraorbital extensions have followed the veins through the anterior lacerated space. Extreme care in dissections is always necessary in order to avoid injury to nerves and blood vessels within the chiasmal and orbital areas.

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FURTHER STUDIES CONCERNING HOMATROPINE CYCLOPLEGIA AND PAREDRINE, WITH SPECIAL REFERENCE TO THE RATE OF ACCOMMODATIVE RECOVERY*

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On May 13, 1940, we reported[1] before this Society the results of our work on a series of 83 patients, showing by strictly comparative tests that the cycloplegic efficiency of homatropine is not enhanced by the use of either Benzedrine sulfate or paredrine. In that study we did not fully investigate the possibility that these drugs may act to hasten the recovery from homatropine cycloplegia. Since then we have studied this question in greater detail in 36 patients, the data obtained comprising the substance of this report. The selection of patients, the plan of procedure, and the techniques of examination and of instillation of solutions have been similar in this work to that previously reported. The following abbreviations are used, as in the first paper:

HA = 5-percent homatropine alone, 2 drops
HP = 5-percent homatropine, 2 drops; 1-percent paredrine, 1 drop
H2% = 2-percent homatropine alone, 6 drops

In addition to the literature reviewed in our previous report, there is a paper entitled "A study of practical cycloplegia," by Wenaas, Evans, and Odom,[2] which appeared in October, 1940, after our present work was well under way. These authors, using the same method of testing residual accommodation, have confirmed our findings (which were not published until March, 1941) of the lack of a synergistic action by paredrine in homatropine cycloplegia. They also state, with out presenting much supporting data, that the addition of paredrine does not hasten the accommodative recovery from homatropine. They, as well as other authors whom they cite, consider that cycloplegia is adequate even with 2 D. of residual accommodation, whereas we believe, with Duane, that more than 1 D. of residual accommodation means the probability of insufficient cycloplegia. We agree, however, with their principal conclusion, to the effect that the dosage of cycloplegics could be more accurately adapted to the differing requirements of individual patients than is usually effected, also with their statement that accommodative recovery from homatropine cycloplegia is not hastened by the addition of paredrine.

As in the first paper, we show in table 1 the characteristics of our clinical material, and in table 2 that in this series, also, evidence is lacking that paredrine actually enhances the cycloplegic effect of homatropine. The results of our studies of accommodative recovery are presented graphically in figures 1 to 10, and by the percentage method in table 3. Tests were made at 2-hour intervals for the first 8 hours, and also at the end of 24 hours.† The artificial-myopia test was employed until the near point for small test type (Jaeger 1 or 4 point) had recovered to 33 cm. or less, after which near-point tests with only the full refractive correction were also made.

Careful study of these charts discloses that the addition of paredrine does not appreciably shorten the duration of ho-

* From the Department of Ophthalmology, Rush Medical College. Read before the Chicago Ophthalmological Society, November 17, 1941.

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TABLE 1

RÉSUMÉ OF FACTORS CONCERNING PATIENTS, EYES, VISION, AND REFRACTION

	Series 1	Series 2	Totals
Number of patients	26	10	36
Number of eyes	52	20	72
Range of ages	12 to 38	15 to 29	12 to 38
	No.	No.	No.
Age 16 to 35	17 or 65%	8 or 80%	25 or 70%
Age 20 to 35	10 or 38%	2 or 20%	12 or 33%
Age under 16	8 or 31%	2 or 20%	10 or 27%
Age over 35	1 or 4%	0	1 or 3%
Color of iris (patients)			
Brown	11 or 42%	2 or 20%	13 or 36%
Blue	15 or 58%	8 or 80%	23 or 64%
Corrected vision (eyes)			
1.2–2	1 or 2%	1 or 5%	2 or 2.5%
1.2–1 to 1.5	51 or 98%	19 or 95%	70 or 97.5%
Refraction factors (eyes)			
Highest meridian over 3.00D.	2 or 4%	1 or 5%	3 or 4%
Highest meridian 1.25D. to 3.00D.	9 or 17%	8 or 40%	17 or 24%
Highest meridian 1.00D. or less	41 or 79%	11 or 55%	52 or 72%
Over 1.00D. cylinder	2 or 4%	0	2 or 2.5%
Variations in refraction findings (eyes)			
Differences of over 0.25D.	4 or 8%	1 or 5%	5 or 7%
Differences of over 0.50D.	0	0	0

TABLE 2

RESIDUAL ACCOMMODATION AT TIME OF REFRACTION TEST (72 EYES)

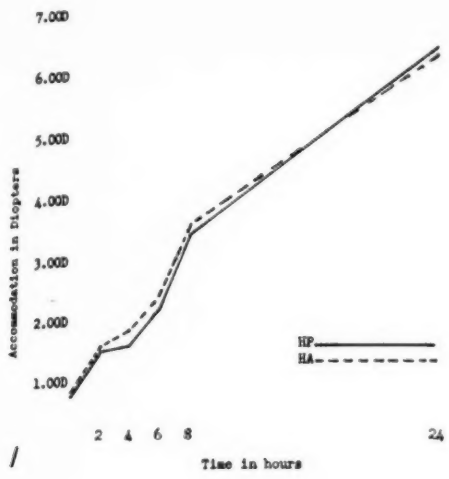
Series 1 (52 eyes)	1.50D.	1.25D.	1.00D.	0.75D.	0.50D.	0.25D.	Totals
Paredrine Homatropine (HP)							
Blue iris	0	0	6	13	10	1	30 eyes
Brown iris	0	3	6	9	4	0	22 eyes
Totals	0	3	12	22	14	1	52 eyes
5% Homatropine alone (HA)							
Blue iris	0	1	9	12	8	0	30 eyes
Brown iris	1	2	8	6	5	0	22 eyes
Totals	1	3	17	18	13	0	52 eyes
Series (20 eyes)							
5% Homatropine alone (HA)	0	0	11	7	2	0	20 eyes
2% Homatropine alone (H2%)	1	2	12	3	2	0	20 eyes

matropine cycloplegia. This was found even in a few cases in which 3 or more drops of paredrine were used instead of one. It is further demonstrated by the percentage figures in table 3, where the differences are too slight to be significantly in favor of paredrine as a factor hastening recovery.

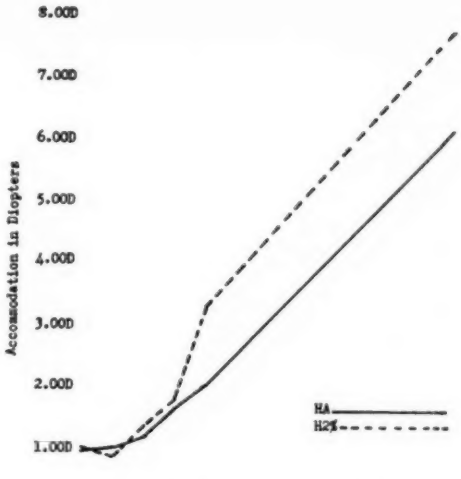
Since practically all of these patients were young, and the majority had low hyperopia, analysis of these factors yields nothing worthy of comment. In fact, patients of these types were selected so as to avoid the introduction of such extraneous factors, and to prevent the inclusion of patients with low accommodative power.

Figs. 1 to 6 (Moncreiff and Scheribel). Graphic presentation of results of accommodative-recovery studies.

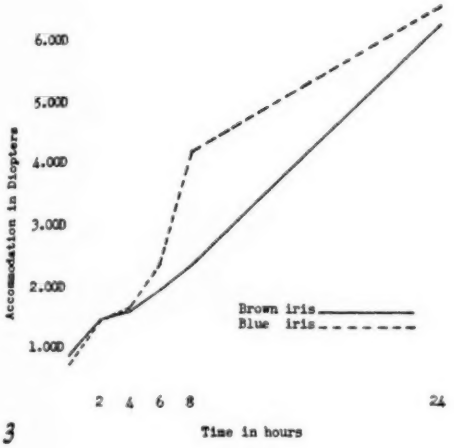
Fig. 1. Series 1. Accommodative recovery (average). Fig. 2. Series 2. Accommodative recovery (average). Fig. 3. Series 1. Accommodative recovery (average). HP. Fig. 4. Series 1. Accommodative recovery (average). HA. Fig. 5. Series 1. Accommodative recovery (average). Blue iris. Fig. 6. Series 1. Accommodative recovery (average). Brown iris.


7.00D
6.00D
5.00D
4.00D
3.00D
2.00D
1.00D
Accommodation in Diopters
HP
HA
2 4 6 8 24
Time in hours

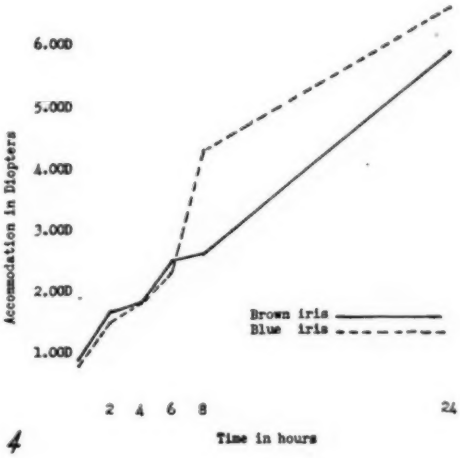
1


8.00D
7.00D
6.00D
5.00D
4.00D
3.00D
2.00D
1.00D
Accommodation in Diopters
HA
H2%
2 4 6 8 24
Time in hours

2


6.00D
5.00D
4.00D
3.00D
2.00D
1.00D
Accommodation in Diopters
Brown iris
Blue iris
2 4 6 8 24
Time in hours

3


6.00D
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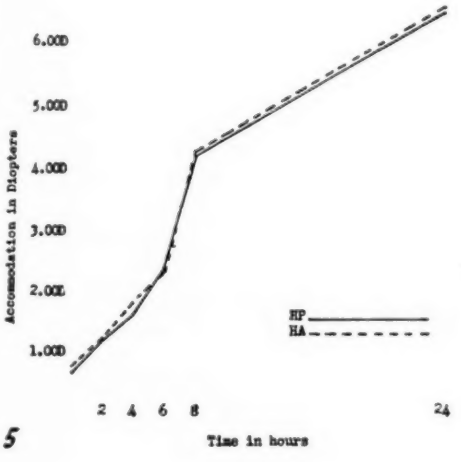
1.00D
Accommodation in Diopters
Brown iris
Blue iris
2 4 6 8 24
Time in hours

4


6.00D
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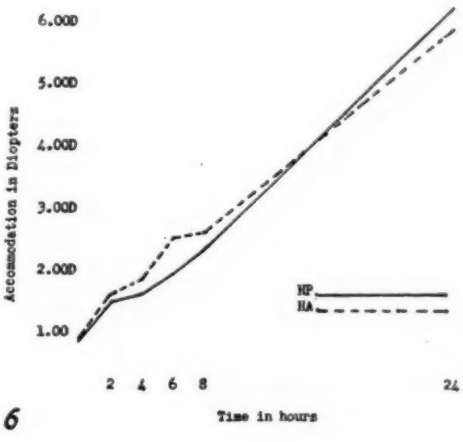
Accommodation in Diopters
HP
HA
2 4 6 8 24
Time in hours

5


6.00D
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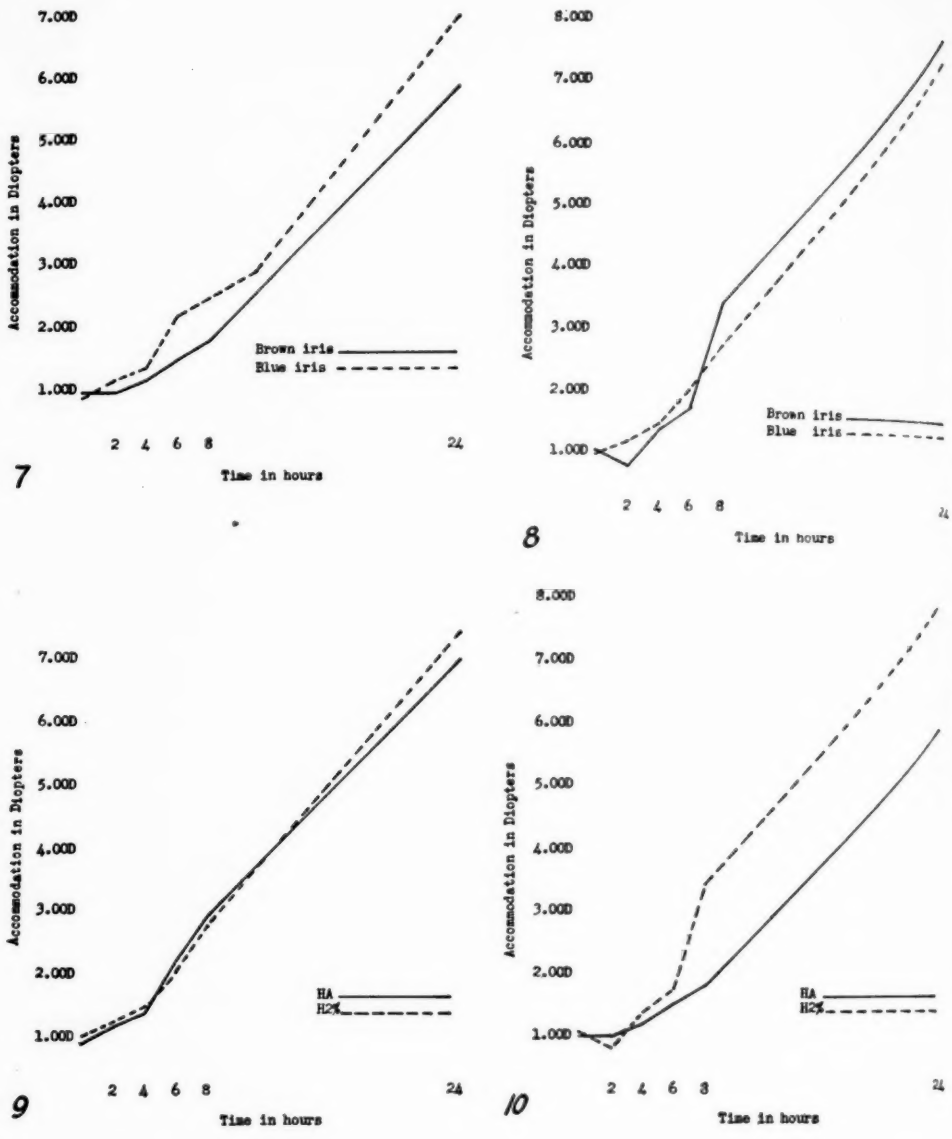
Accommodation in Diopters
HP
HA
2 4 6 8 24
Time in hours

6



Figs. 7 to 10 (Moncreiff and Scheribel). Graphic presentation of results of accommodative-recovery studies.

Fig. 7. Series 2. Accommodative recovery (average). HA. Fig. 8. Series 2. Accommodative recovery (average). H 2 percent. Fig. 9. Series 2. Accommodative recovery (average). Blue iris. Fig. 10. Series 2. Accommodative recovery (average). Brown iris.

The factor of iris color, which in about two thirds of the cases was blue, has not proved to be especially significant in regard to accommodative recovery at the end of 24 hours.

In 10 patients, an additional study was made of the comparative efficiency and of the rate of recovery between 2 drops of 5-percent homatropine and 6 drops of 2-percent homatropine. While the number

of patients is small, the comparison did not show the 2-percent homatropine dosage to be the more efficient (rather the contrary), nor was there a more rapid recovery from the 5-percent homatropine (as one might anticipate), but rather the reverse.

Summary and Conclusions

Further studies on carefully selected patients show good clinical evidence that the rate of recovery from homatropine cycloplegia is not hastened by the action of paredrine. Additional evidence is also presented that the efficiency of homatropine cycloplegia is not enhanced by paredrine.

TABLE 3

ACCOMMODATIVE RECOVERY IN PERCENTAGES OF TOTAL ACCOMMODATION (AVERAGE)

Time	Series 1 (52 eyes) 10.35D. Average Accommodation		Series 2 (20 eyes) 9.90D. Average Accommodation	
	HP	HA	HA	H2%
8 hours	32.6%	34.2%	19.8%	32.7%
24 hours	61.5%	60.4%	61.6%	78.2%

In a series of 10 patients, the action of 2 drops of 5-percent homatropine was found not to be superior to that of 6 drops of 2-percent homatropine with regard to either the efficiency of the cycloplegia or the rapidity of recovery.

58 East Washington Street.

55 East Washington Street.

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MYOPIA*

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By the refraction of an eye is meant the relationship that exists between the combined power and regularity of the refractive system and the length of the eye. Depending on these conditions, we have either emmetropia or ametropia. An eye may be emmetropic or ametropic regardless of whether it is healthy or diseased.

Throughout life certain changes take place normally in every healthy eye. The greatest are those in the shape of the crystalline lens and in the length of the eyeball, which occur during childhood and adolescence and which, sometimes, alter the refraction to a considerable degree.

These changes are natural and occur in perfectly healthy eyes; but alterations of the power and regularity of the dioptric system, and the relationship between it and the length of the eye can be disturbed also by disease or trauma. The ametropia that is induced by disease is termed pathologic. Hence we have such designations of ametropia as pathologic astigmatism, pathologic or malignant hypermetropia, and pathologic, pernicious, or malignant myopia. The ametropia, of course, is not the disease; it is the result of the changes that have taken place in the eye as a whole because of the disease. Every ophthalmologist will agree with the general statement that a refractive error is not a disease; nevertheless, choroiditis, retinitis, vitreous opacities, or detachment of the retina are spoken of frequently as complications of myopia. The occurrence of such conditions is in itself evidence of disease, whether the eye happens to be hypermetropic, astigmatic, or myopic. No ophthalmologist confuses typical hypermetropia or astigmatism with the so-called pathologic or malignant forms, but a great many seem to see no difference between typical myopia and a diseased eye that results in or happens to be associated with myopia. One would have to search quite diligently for references under the heading of pathologic hypermetropia or pathologic astigmatism, but the literature teems with articles on pathologic or so-called malignant or pernicious myopia. The reason for this is that many really great ophthalmologists believed, and still do believe, that every myopic eye is potentially a diseased eye. Even Donders[1] said that every myopic eye is unsound.

An analysis of the vast amount of literature on the subject leaves no doubt in one's mind that whereas most ophthalmologists rightly agree that typical myopia and pathologic myopia are separate and distinct conditions, there seems to be a wide diversity of opinion regarding the line of separation between the two fundamental classes. Some, like Fuchs[2] and Gullstrand,[3] would classify the mild degrees of myopia as typical and the high degrees as pathologic, regardless of whether or not there is evidence of disease. Others would divide them according to the rate of increase, placing the stationary or slowly progressive cases in the benign class and the more rapidly progressive ones in the pathologic or malignant class. But many others, including myself, believe that myopia is simply an anomaly of refraction as long as there are no symptoms of disease and normal visual acuity can be obtained with proper correcting lenses.

I cannot agree with Donders's statement that just because an eye is myopic

* Read before the American Academy of Ophthalmology and Otolaryngology, at Chicago, October 20, 1941.

it must be considered unsound. Neither can I agree with the opinion of Gullstrand[3] that the myopic are to be considered as people who must pay for civilization by being disabled, nor with Keith,[4] who wrote that we must regard myopia as a structural disharmony occasioned by the conditions that civilization has entailed on us. Theory after theory has been proposed to show that myopia is a primary disease and to account for the incidence and causation of any case of myopia, typical or pathologic.

The vast number of statistics on the distribution of myopia would lead us to believe that the more civilized a nation, the more myopia; but, with regard to the distribution, one may find almost anything he desires. Ware,[5] as early as 1812, made the remarkable claim that among nearly 10,000 foot guards not half a dozen privates were discharged, nor had a dozen been rejected on account of myopia in the space of 20 years; but that on the contrary, in one college at Oxford, 32 myopic students were found out of 127 examined. But Tscherning,[6] in 1883, found the distribution of myopia among 7,000 Danish conscripts to amount to 7 percent in laborers and peasants. Doyne[7] said that typical myopia is much more common among the educated and in such occupations as require near work. But pernicious myopia is just as frequent among the illiterate as in other classes. Galloway[8] found many cases of high myopia in farm servants. Kirk[9] considered myopia essentially a disease of town dwellers and of industrial life. In a series of 300 agricultural laborers he found only one case of axial myopia above one diopter. Thompson,[10] however, found 17.6 percent of myopia in children of the city and 24.2 percent in rural children. The association of myopia with poor hygienic conditions and faulty illumination during early life has been emphasized often, but Lawson[11] found the percentage of myopia actually 5 percent more in the better-lighted and -ventilated schools.

Almost every conceivable reason has been offered for the causation of myopia, a refractive error: overaction of the ciliary muscle by excessive close work and accommodation; stretching of the choroid by pulling of the ciliary muscle; deficient effect of the action of the extraocular muscles; increase of the intraocular pressure with weakness of the sclera; cardiovascular disease; pulling on the optic nerve by gravitation; lack of calcium; increase of blood in the orbital tissues and the eye; disproportion of the size of the retina to the eyeball; through the turnstile of astigmatism; bending of the upper part of the body; congestion by manual labor; shape of the orbits; lack of vitamins; glandular dysfunction; measles, whooping cough, and bronchitis; obstruction of lymphatic drainage; prenatal and inherited weakness; and many others, including of course, tuberculosis, syphilis, and sinus disease. Junius[12] said that light, a physical and chemical stimulus of the retina, may become a malign influence in the predisposed, and Incze[13] associated myopia with flat feet.

Some of these theories have many adherents, some have a few, and many have only one, the proposer. Every theory that has ever been offered can be disproved by argument, clinical experience, or experimental investigation, and mostly this has been done by the author of a hypothesis of his own.

In the absence of any symptoms of disease, myopia, regardless of degree, is a simple error of refraction due merely to the fact that the retina lies behind the posterior principal focal plane of the dioptric system of the eye. There is no known way to prevent, arrest, or cure it.

The terms pathologic, malignant, and pernicious myopia should be no longer

used. They indicate some disease that either occurs in a myopic eye or that happens to result in myopia. These eyes may eventually go on to blindness; but because of the disease, not the myopia. So-called pathologic or malignant myopia is pathologic from the beginning. Never have I had nor have I been shown a case of simple myopia in a healthy eye that developed into the so-called malignant type.

The management of myopia either in a child or in an adult depends on whether we are dealing with a simple anomaly of refraction or with a disease. The term "progressive myopia" has no meaning. With few exceptions the refraction increases during the growing age and in such cases myopia in a child is naturally progressive, more or less. The process may or may not be affected by disease. If, in a case of choroiditis, myopia happens to be associated, a diagnosis of myopia most likely will be made, otherwise the associated refraction is disregarded. Of course, an eyeball may lengthen as a result of disease, but we certainly see the same conditions—uveitis, posterior staphyloma, choroiditis, detachment of the retina, and others—that are so often called complications, and believed to be characteristic, of myopia, occur in emmetropic, hypermetropic, or astigmatic eyes.

Simple myopia regardless of whether it is low, medium, or high, provided there is no evidence of disease and the absolute visual acuity is normal, is merely a form of ametropia. It is no more serious than hypermetropia or astigmatism, except that it is likely to increase as the globe grows larger with the rest of the body. Myopia in infancy is rare. It seldom becomes manifest until the seventh or eighth year. Its progression, therefore, corresponds to the school age, hence the misleading term "school myopia."

It has never been shown that anything we can do will prevent the onset or arrest the progress of typical myopia or any other refractive error. We have no more means of controlling the growth of the eyeball than we have of regulating the growth of any other feature of the body. Certainly in my experience it makes no difference, with regard to the ultimate results, whether glasses are worn or not. Neither does it make any difference whether the patient does close work or not. The region of accommodation is small in myopia; but, generally speaking, the power is just as great in myopia as it is in hypermetropia. I do not believe that there has ever been any evidence to sustain the theory that typical myopia can be produced, or the increase of an already existing typical myopia be influenced, by close work. The very men who claim this advocate a full correction, to be worn constantly, and so compel the corrected eye to use still more accommodation than if it were left uncorrected. Even removal of the crystalline lens has no effect on the progress of myopia. Accommodation is exercised to a less degree in uncorrected high myopia than in uncorrected low myopia, but the progress is as great or greater in high grade as it is in low grade myopia. In the case of unequal myopia in which, uncorrected, no accommodation whatever is needed for close work, the more myopic eye often progresses at a greater rate than the better one. Also, if this theory had any value we should expect that the process would slow down in the case of a child who has gone over from hypermetropia to myopia, when less accommodation becomes necessary.

Neither is convergence nor the position of the head a factor in the production of myopia. Myopia associated with convergent squint is extremely rare. On the contrary it is quite commonly associated with divergence, but it seems unreason-

able to believe that the myopia is the result of the divergence or of an exophoria. It is much more reasonable to suppose that divergence can be a physiologic result of the myopia. Myopia cannot possibly be either arrested or reduced by the use of prisms.

Nearly all children have the same way of reading a newspaper or book. They lay it on the floor and lie down with their heads hanging within a few inches from the object. Here we have the faulty position of the body, excessive accommodation, overaction of convergence, and poor light all at the same time. Children do this day after day for years. Also, nearly every child has had measles, whooping cough, or bronchitis at some time. If any one or more or these were factors in the production of myopia we certainly should have much more of it.

In my experience there is no myopic type any more than there is a hypermetropic or astigmatic type. I have observed myopia with equal frequency in long or broad heads, in short or tall, in healthy or unhealthy, in delicate or robust persons.

For the purpose of obtaining the best visual acuity it is probably good to give a full correction in myopia. Also, examinations should be made at comparatively short intervals of time in order to keep the correction up to the increasing refraction. But, in my opinion, it is useless to order restrictions of any kind with regard to the use of the eyes of a myopic child. One would hardly think of such a thing in the case of a child of five or six years of age with emmetropia, although this child surely can be expected to become myopic. There is no reason why a myopic child should not be allowed to go without his glasses whenever they interfere with his activities. It is most important that parents should not be made to believe that the child is afflicted with a disease that might result in blindness. No one goes blind from a refractive error.

Because myopia is often associated with disease, particularly low-grade uveitis, every method of examination should be made to detect the slightest evidence of changes in the cornea, iris, lens, or fundus. These are the conditions that might be due to some general disease—deficiency in certain nutritional elements, lack of vitamins, lack of calcium, faulty endocrine secretions, or any of the many other etiologic factors suggested as the cause of myopia, but which really are the cause, directly or indirectly, of a chronic disease of the eyes. The ophthalmologist is required here to treat a disease, not an ametropia. It is up to him to treat it as he sees fit—restrict the use of the eyes, seek a possible cause, and give whatever medication he thinks is indicated. But this disease cannot be cured by wearing concave glasses or prisms, exercise of the extraocular muscles, massage of the eyeballs, or any of the many other methods in use at the present time.

1930 Chestnut Street.

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Discussion

Dr. E. C. Ellett (Memphis): It has long been known that the eyes of an infant are hyperopic and that this tends to decrease as the child grows, but usually some degree of hyperopia persists, though it may continue to decrease until the child is grown and may pass over into myopia. This tendency is recognized in the recent publications from the U.S. Naval Academy, which are to the effect that a student who has 1 diopter of hyperopia or less is apt to become myopic by the time of his graduation and will not be suitable material for a naval officer.

The idea formerly prevailed that all myopia tended to progress indefinitely and end in disaster, but we know that there is a limit to the amount of increase and that the usual type is what Parsons called "developmental myopia," and which ran about the following course:

A child, who has had no previous complaints, and who possibly on several occasions has been found to have normal vision, begins to complain at about the age of 8 or 10 of poor distance vision (blackboard, movies, and other places) and is found to have myopia of 1 diopter or so. In spite of everything that is done, or if nothing is done, the myopia increases, until the age of about 20, to the extent of 3 or 5 diopters, and then becomes stationary. In answer to the distress of the parents over the child's wearing glasses, especially if the patient is a girl, we try all the things we have heard of, including glasses, to arrest the progress, and they have not the slightest effect. That does not mean that all cases progress, for many do not, and many only progress to 2 or 3 diopters and then stop. Of the many means that are advised to alter the course of the disease, if we may call it that, the following may be mentioned:

1. Glasses.
2. Restricting or prohibiting near work.
3. The use of adrenalin.
4. Atropinization of one eye.

In attempting to evaluate these plans I have analyzed the records of 300 private patients, not selected, but taken as they came, who have been under observation from 3 or 4 to 40 years, and my opinions are based on a study of their records.

1. *Glasses.* Most textbooks advise giving a full correction, or —.25 D. sph. more than the full correction, for constant wear. It is helpful as far as the vision is concerned, but my personal experience is that it has no effect in preventing the increase of the myopia. I can report that under this plan some patients did not show any progress in their myopia, but an equal number who wore glasses occasionally or less often showed the same tendency to remain stationary. A few typical cases are as follows:

TABLE 1

ILLUSTRATING TYPICAL DEVELOPMENTAL MYOPIA

	Age First Test	First Test	Myopia Began	After 2 Years	After 5 Years	After 10 Years	After 20 Years
1	8	+ .50 D. sph.	12 −1.50 D. sph.	−2.00 D. sph. −1.50 D. sph.	−2.25 D. sph. −1.75 D. sph.	−3.00 D. sph. −2.75 D. sph.	
2	10	−3.50 D. sph.	8	−4.00 D. sph. −4.50 D. sph.	−5.00 D. sph. −5.50 D. sph.	Same	Same
3	9	EM	16		−1.75 D. sph. − .50 D. sph.	−2.25 D. sph. −2.50 D. sph.	No change after 19.

TABLE 2

ILLUSTRATING COURSE OF MYOPIA OVER MANY YEARS

	Age First Test	First Test	Myopia Began	After 5 Years	After 30 Years	After 45 Years
1	16	−4.50 D. sph. OU	14	−4.75 D. sph. OU	−5.00 D. sph. −4.00 D. sph.	
2	7	−4.00 D. sph. OU	7	−7.00 D. sph. −8.00 D. sph.		−8.50 D. sph. −9.00 D. sph.

TABLE 3

PATIENTS USING EPINEPHRINE FOR 2 YEARS

	Age First Test	First Test	Myopia Began	After 2 Years	After 5 Years	After 10 Years
1	6	− .50 D. sph.	6	−1.00 D. sph. − .75 D. sph.	−1.75 D. sph. −1.25 D. sph.	−3.25 D. sph. −3.00 D. sph.
2	14	EM	16	− .50 D. sph. and cyl.	−1.00 D. sph. and cyl.	−1.00 D. sph. and cyl.
3	8	−1.50 D. sph.	7	−3.00 D. sph.	−4.25 D. sph. −3.50 D. sph.	
4	16	−1.25 D. sph. −1.50 D. sph.	15	−2.75 D. sph. −3.00 D. sph.	−4.25 D. sph. −3 .75 D. sph.	

If the patient is a girl, you may be sure that she and her mother will cöperate to have her leave the glasses off on social occasions, to which I see no objection.

I have under observation a few women whom I have known for 40 years or more and who have worn their glasses very little or almost not at all, and, at the end of 40 years, these women of 60 or past are no worse off than are others with the same type of trouble who have worn their glasses constantly. In this, as in other plans for treating myopia, one is struck with the analogy to one's experience in treating cataracts. Some cases progress and some do not, and it is difficult to say if any treatment has any effect in either condition.

The question of the value of contact glasses in treating and preventing the progress of myopia is still undecided. My feeling is that since they do not and should not make pressure in the eye, they could not do any good. We hesitate to advise them until the myopia is stationary.

2. *Restricting or prohibiting near work.* It is a common observation that myopic children are bookworms and find their main source of entertainment in reading. It is impossible to suspend accommodative acts effectively, because at the table, in conversation, in brushing the hair, they depend on the accommodation. If a child is taken out of school, how long must he stay out? What assurance can we give that the myopia will be arrested by such a plan? I have met this angle of the

situation by advising that the eye work be limited to necessary school work, and encourage the child to entertain himself by other means—such as, swimming, skating, dancing, and outdoor sports—but such advice is not often strictly followed, nor do I know that it helps if it is.

3. *The use of adrenalin.* Meyer Wiener has advised the use of 1:1,000 epinephrine three times a day as a means of preventing the progress of myopia, and Gradle and others have reported favorably on it. In this series of 300 cases, epinephrine was prescribed for 103 children. It was not used faithfully in many of them, and I would consider that its use for less than a year was not a fair test, but in those who tried it that long I have not observed more than an occasional cessation of the increase in the myopia. I can report quite as many who received no treatment or only glasses and hygiene and in whom the myopia did not progress. My experience with it, therefore, is not encouraging.

4. *Atropinization of one eye.* This plan was suggested and the details explained by W. H. Luedde (Amer. Jour. Ophth., 1932, July). It harks back to an observation by Donders to the effect that watch makers, who use a monocular loupe, do not show a progress in myopia. The suspension of the accommodation in one eye by atropine abolishes or diminishes the effect of convergence, which many observers think is the potent factor in progressive myopia. It may explain some cases, and in these the method is worth trying. Luedde reports favorably on it. I have suggested it to several patients, but so far have not been able to give it a fair trial. Convergence does not explain all the cases, nor does Luedde claim that it does. For example, a boy of 10 was seen in May, 1941, having lost the vision in the left eye in September, 1940, as the result of an accident. He was seen at the time of the injury by Dr. Donald O'Rourke, of Denver, who did not measure the refraction of the right eye but noted vision of 6/6, which certainly excluded myopia at that time. In May, 1941, a test under homatropine showed vision 6/15 and myopia of 1 diopter.

5. Such general tonic and other treatment as is indicated should be given, and some children need certain vitamins, especially vitamin A, but there is nothing specific in their use as far as myopia and its progress are concerned.

The myopia that is congenital or that becomes high at an early age is quite a different matter and is not, I take it, under consideration now. I have recently seen a child less than a year old with myopia of between 20 to 30 diopters, as measured with the ophthalmoscope, and others of less, although almost as high, degree.

My experience and the opinions resulting from it agree very closely with those of Dr. Cowan. It does seem to me, however, that myopic eyes are particularly susceptible to low-grade uveitis, of which the commonest manifestation is vitreous opacities, but I am not sure which is the cart and which is the horse. They are also, as a group, subject to more and greater changes in their refraction than other people are, and for this peculiarity I do not know a satisfactory explanation.

DR. ARTHUR ALEXANDER KNAPP (New York): Dr. Cowan's remarks are very interesting, but it seems that we have varying results, particularly with vitamin therapy. Dr. Cowan states that myopia is a localized condition. It is known that myopia of any type is not purely a localized refractive error, but is a part of a systemic status just as there is a tubercu-

lous status. This was recognized years ago.

In the résumé of his paper this afternoon, Dr. Cowan states that in the light of our present knowledge neither myopia nor any other refractive error can be controlled any more than a separate growth of any other feature of the body can be controlled. It might be said that following the administration of irradiated ergosterol and mineral-mixture tablets, not only the eye but also the whole body may undergo a change.

Briefly, I would like to outline in just a few moments the work that we have done in the Department of Pharmacology at Columbia University. Blackberg and I have carried on experimental work with dogs and rats, inducing what we felt was myopia due to a diet deficient in vitamin D and calcium. We enucleated some eyes, and under the microscope the pathology primarily found was a generalized edema of the fibrous tunic, which caused a weakening of this outer coat. Subsequently, at the New York Eye and Ear Infirmary, I carried on further research work on patients with anterior curvature and progressive axial myopia. Careful examinations of their eyes revealed less axial myopia in some and improvement in all cases of keratoconus. Accurate measurements of plaster-of-Paris casts of these eyes were made by Prof. Carlos De Zafra, of the Department of Engineering, and Prof. Louis Granath, of the Department of Physics, both of New York University. They carefully measured these casts, and their findings corroborated my clinical experience. Definitely they showed that the eyeball may actually shrink following the administration of vitamin D and calcium.

More than 70 casts of both types of myopia have been measured (I have brought four with me), and the improvement is macroscopic. I will leave them here on the desk for your observation if you desire to see them after the meeting.

Furthermore, as to progressive myopia, more than 50 percent of the patients showed that they had either less myopia or it remained stationary following vitamin-D and calcium therapy. Supplementary reports, unpublished, corroborate these findings.

This afternoon I should also like to show you that the fundus of the eye, or the posterior segment, may shrink. The fundus picture which you will see is that of a woman of 39 who had myopia of 12 diopters and who, after the administration of vitamin D and calcium, showed a shrinkage of the posterior segment of the eye. The photographs were taken under identical conditions at the New York Eye and Ear Infirmary by the same photographer. It may be stated that the measurements were made by Mr. Host, of the American Optical Company, shortly before this meeting.

(Slide.) This is the first slide, taken before therapy. The vertical distance from this fixed point to this (the superior and inferior margins of this central staphyloma) was measured at 3 feet and 1 inch. Using these fixed points (the crossing of the vessels at the superior border of the disc and the crossing of these vessels in the macular area) the horizontal distance was 4 feet, 2.5 inches.

(Slide.) This is the later slide taken after six months' treatment with vitamin D and calcium. The vertical distance between these two points now measures 2 feet, 11.5 inches, whereas the horizontal distance measures 4 feet, 0.5 inch, a decrease in the vertical diameter of 1.5 inches on the screen, and in the horizontal diameter, a decrease of 2 inches, showing that both the vertical and horizontal diameters of the posterior segment of the globe may shrink following treatment with vitamin D and calcium.

It should be borne in mind that no matter what classification one arbitrarily makes, it is not reasonable to expect any type of untreated myopia to be reduced. By the casts presented to you and the fundus photograph projected on the screen, it may be concluded that myopia actually may be reduced by a shrinkage of both the anterior and the posterior segments of the globe following vitamin-D and calcium therapy.

Dr. Sanford R. Gifford (Chicago): I want to congratulate Dr. Cowan for his taking this courageous and very unpopular position. Everybody knows that parents come to us with children who have something the matter with them, and they all want us to do something for these children. They want us to cure the condition, and anyone who offers to do anything for myopia at once puts himself in a position of tremendous advantage over his colleagues in the same community. I think the only thing that we as honest ophthalmologists can do, however, is to face the facts and tell these parents what the facts are about myopia. I think that Dr. Cowan has told us what to do, and it is essentially the whole truth in the matter.

One might speak of the vitreous opacities or of some of the degenerative changes of the fundus in myopia, but one also knows that these also occur as senile changes, and Vogt has shown that all these changes can be seen in senility. I have taken somewhat the same position in the chapter on refraction in my textbook, and I should like to put my neck out just a little farther and take up the question of sight-saving classes. The treatment of children suffering from simple myopia by placing them in sight-saving classes seems to me to be a total mistake and to disregard entirely the real purpose of these sight-saving classes, which is to afford a place for children who cannot see well, to study under conditions in which they can see. It seems to me that there is no sense in putting myopic children in sight-saving classes, and we ought to admit it.

There is just one suggestion in the management of the myopic that I might make, and that is it has often seemed advisable to give them less than their full correction for reading. They often complain of asthenopia when wearing their full correction.

I should like to ask Dr. Cowan one question. We tell these parents, at least I tell them, that the only thing they can do for their children is to have them lead healthy lives. If they need vitamins, give them vitamins. If they have rickets, give them vitamin D. Nobody has ever proved, it seems to me, that in a large number of myopic patients there is a deficiency of vitamin D or any other vitamin any more than there is this deficiency in the average population. But we always tell these parents that we can expect the progress of myopia to stop at about the time the child reaches his full growth. Occasionally we see patients in whom that progress seems to continue for some time past the age of 21. I should like to know what Dr. Cowan's experience has been in that respect.

Dr. Alfred Cowan (Philadelphia): The remarks of Dr. Ellett and, to a great extent, those of Dr. Knapp, back me up in my contention. There is no doubt that we find a number of myopic patients with vitreous opacities and other lesions; but we know that vitreous opacities, whether we see any other lesion in the eye or not, should be considered an indication of some diseased condition, past or present. It is not myopia *per se*, but a disease associated with myopia. We certainly see vitreous opacities in other forms of ametropia. When Dr. Knapp speaks of anterior-

curvature myopia I presume that he means keratoconus. That is not what I consider simple myopia. It is keratoconus. He says that 50 percent will remain stationary with his method of treatment. Now I do not say that his method of treatment is not good. I have not carried out that method to the extent he has and, therefore, he is in a better position than I to make positive statements; but I do not consider 50 percent of arrests such a great result. Probably 50 percent of the myopias in our general practice remain stationary without any treatment at all. I do not know the number, but we do have many that remain stationary.

The eye that Dr. Knapp showed on the slide is, of course, a diseased one—choroiditis. Treatment of such a condition has nothing to do with treatment of myopia.

Dr. Gifford was my nicest discussant. His was the kind of discussion I like. I am glad he brought up the subject of sight-saving classes. I have been fighting them for years. I feel that this term should be dropped and the term "low-vision class" substituted since they do not save sight. Children who cannot be made to see well should be placed in low-vision classes and, for them, such classes are useful. I know for a fact that in Philadelphia, in the so-called sight-saving classes, they take, or have taken, children with perfectly normal visual acuity with glasses, if they happen to be myopic more than 5 or 6 diopters or if the ophthalmologist who is managing the case has the idea that they must not use their eyes for ordinary work. The child is placed with children having low vision, and so a child who is not handicapped at all is given a certain complex. Because the ophthalmologist feels that the child might lose his sight some day, he forbids the use of the child's eyes. This is sight saving. What is he saving the child's sight for? Why shouldn't a person use his sight while he has it?

Some of these children have even been admitted to institutions for the blind. The object is to teach them Braille or the ways of a blind person because they might some day become blind. It would be much better to educate such children to the greatest extent possible while they still see.

I explain to the parents of a child with simple myopia that the child has myopia and that myopia is nothing but a refractive error. He will always need glasses; but most people wear glasses nowadays, and a person's eyes are just as good as they can be made to see with glasses. I emphatically state to the parents that their child's eyes are not diseased.

I think that parents should not believe nor should they lead the child to believe that because he needs concave lenses he is afflicted.

As far as the progress of the condition is concerned, it increases, sometimes up to 25, 30, and even up to 35 years of age; but just as long as there is no disease it does not matter.

ANOMALOUS PROCESSES OF THE CILIARY TYPE ON THE POSTERIOR SURFACE OF THE IRIS*

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The anomaly to be described consists of processes attached to the posterior surface of the iris. They closely resemble the ciliary processes from which they are separated by normal iris tissue. In 1907, Gallenga[1] described one case and in 1935 Reese[2] reported six cases of this anomaly. Reese found it very frequently and remarked on the rarity of its description in the literature apart from the report of Gallenga and some remarks by Eisler[3] and Lauber.[4] Reese noted that in eyes completely sectioned, anomalous processes were found in six out of eight cases. While I have not found it in this ratio, it certainly is not rare.

Microscopic study of a large series of globes disclosed 26 specimens with this anomaly. In none was there any evidence of injury or disease that could have produced this picture. In my series, the ages were from birth to 67 years, about equally divided between the sexes. In some eyes, only one anomalous process was found, while in others more were seen. In one completely sectioned eye, five separate anomalous processes were found attached to the iris.

In these 26 eyes and those described by Gallenga and Reese, the anomalous processes were found near the root of the iris. In the usual globe, most of the iris conforms to the classic structure, but it is not uncommon to find in the periphery of the iris an occasional anomalous ciliary process.

These processes are about as long as the average ciliary process. Their diameter varies, but it is usual to find them in 5 to 20 consecutive sections, each about 10 to 15 micra in thickness. It is obvious that the chances of seeing one of them are not great unless many sections of each eye are examined.

The anatomy of the anomalous processes closely resembles that of the ciliary processes, except that they are attached to the iris. The change in the epithelium over the iris and the anomalous processes is interesting. This is best studied by starting with the epithelium on the ciliary body and tracing it forward. The ciliary body is covered by two layers of epithelium, the outer clear, the inner pigmented. In following them forward they are seen to change at the iris, so that the outer clear layer becomes the deeply pigmented cells of the posterior surface of the iris. The inner pigmented layer of the ciliary body forms the dilator muscle of the iris. At the anomalous ciliary process on the iris, there is reversion of the layers, so that the outer pigmented layer of the iris again becomes clear, and the dilator muscle becomes interrupted and transformed into pigmented epithelium similar to that of the other ciliary processes.

The dilator muscle is interrupted at the outer rim of the attachment of the anomalous process to the iris, and so across the base of the process, there is an island of iris without dilator fibers. This break in continuity of the dilator fibers is important, for it must cause defective dilata-

* From the service of Dr. Robert K. Lambert and the Garfunkel Eye Laboratory at Montefiore Hospital. I am indebted to Dr. Bernard Samuels for his advice, and for his kindness in permitting the examination of many additional specimens at the New York Eye and Ear Infirmary.

Figs. 1 and 2 (Gartner). Ciliary-type processes on the posterior surface of the iris. Figure 1, isolated process; figure 2, branched process.

tion of the iris. The transformation of the pigmented epithelium of the iris to the clear epithelium of the anomalous process occurs at a little distance from the base of the process, usually about one third of the way to the tip.

Within the anomalous processes the tissue is similar to that of normal ciliary processes. There are a mass of connective tissue, small blood vessels, and clumps of pigment.

The anomalous processes appear in various formations with considerable variation and blending of one type into another. Classification according to their outline may be made as follows:

1. Isolated single processes that are separated from the ciliary processes by normal iris tissue (fig. 1).

2. Branched processes that are similar to the first group, except that the main process has budlike branches that extend out in various directions (fig. 2).

3. Processes with bridges of tissue that connect with the ciliary processes arising from the ciliary body (fig. 3).

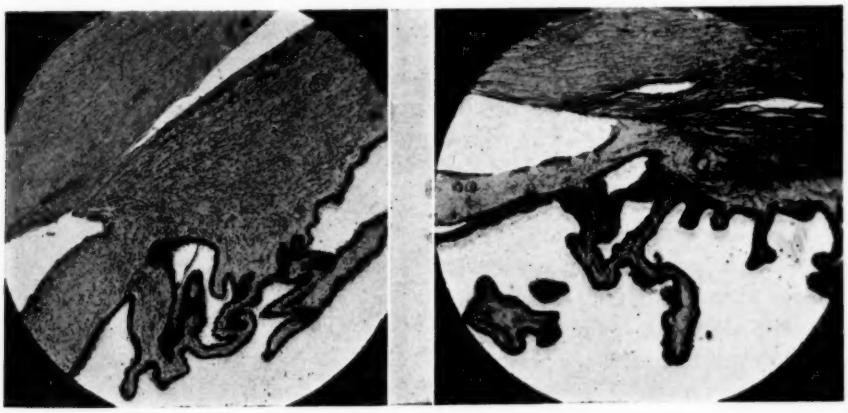

Fig. 3 (Gartner). Processes on the posterior surface of the iris connected to the ciliary processes by bridges.

Fig. 4 (Gartner). Fetal eye of about four months, demonstrating early development of the ciliary processes.

There is every reason to believe that the anomalous processes have the same function as the ciliary processes in the formation of aqueous. Thus a portion of the iris may take part in secreting aqueous.

Normally the root of the iris at its attachment to the ciliary body is a little thinner than the rest of the iris. In several eyes exhibiting isolated ciliary processes on the iris there was abnormal thinning of the root of the iris. Associated with the anomalous processes there is usually an increased blood supply to that part of the iris. In some cases there is displacement of the greater arterial circle to the iris root, as in figure 1. It can be readily appreciated that in such eyes iridectomy would greatly increase the amount of hemorrhage.

If eyes having such anomalous processes should undergo an iridectomy, more hemorrhage and postoperative reaction could be expected. Should the anomalous process be bridged to the ciliary processes, as was the case in figure 3, an iridectomy to the root of the iris would be difficult to perform, would be certain to tear through ciliary tissue, and add complicating factors to the operation. This may well account for some of the complications following iridectomy. Injury and infecion of the anomalous processes might cause sympathetic ophthalmia.

The basis for this anomaly must be sought in an embryologic study of the eye. The ciliary body and iris first make their appearance in the third month of fetal life. The iris develops as an outgrowth from the ciliary body. Soon after the development of the iris there appear folds on the inner surface of the ciliary body and to some extent on the iris near the ciliary body (fig. 4). From these folds the ciliary processes develop. Ciliary folds on the iris are frequently seen until the ninth month of fetal life. At the time of birth the ciliary processes on the iris normally vanish (Eisler,[3] Gallenga,[1] Axenfeld,[5] Lauber,[4] Wolfrum,[6] Reese[2]). There is, however, considerable variation, as is seen in figures 1 to 3, where ciliary processes on the iris have persisted. The anomalous processes are apparently fetal remains of tissue which failed to disappear in the normal manner.

Fig. 5 (Gartner). Eye of a rabbit with ciliary processes attached to the iris.

Ciliary processes on the posterior surface of the iris are normally found in a number of animals. In the rabbit, the ciliary processes arise from the ciliary body and are attached to the iris (fig. 5).

Summary

Twenty-six cases are reported of anomalous processes on the posterior surface of the iris. They closely resemble the ciliary processes, from which they are separated by normal iris tissue. The anomalous processes are fetal remains of tissue which usually disappear by the time of birth. Their presence can assume practical importance in complicating an iridectomy, in as much as they may cause increased hemorrhage and postoperative reaction.

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DISPLACEMENT OF CILIARY PROCESSES TO THE POSTERIOR SURFACE OF THE IRIS*

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The textbook picture of the ciliary body at the root of the iris is rather standardized. It shows an abrupt transition from the ciliary body to the iris. At the point of attachment, the ciliary processes end, and the epithelium covering the ciliary body changes to that of the iris. That is the way it is pictured in the texts by Fuchs,[1] Salzmann,[2] Duke-Elder,[3] Parsons,[4] May,[5] and Gifford.[6]

Actually this arrangement is found in about three fourths of the eyes examined. In the other fourth there is a variable amount of forward displacement of the ciliary processes to the posterior surface of the iris. Only in the article by Bernard Samuels[7] in Berens's "The eye and its diseases" is there illustrated this feature of the anatomy at the iris root.

Very frequently the ciliary processes are arranged as in figure 1, A to F. They are distributed only partially over the ciliary body and extend for another one to two millimeters over the posterior surface of the iris beyond its root. This displacement of ciliary processes to the iris is not the same in all meridians of any one eye. In some portions there may be considerable displacement, in other parts less, or none at all.

The iris consists essentially of stroma, dilator muscle, and pigment epithelium. These layers do not always arise together at the same level. Iris stroma begins rather precisely at the root of the iris. The vessel layer of the ciliary body and its epithelium may continue on the posterior surface of the iris for some distance. They end where the ciliary processes end. At that point there first appear the dilator muscle and the pigment epithelium of the iris. When, therefore, the ciliary processes are displaced for a few millimeters on the posterior surface of the iris, the posterior layers of the iris begin a few millimeters beyond the root, causing to that extent a deficiency of the dilator muscle. This must result in defective dilatation of the iris.

The ciliary processes are highly vascular. When displaced to the posterior surface of the iris they carry with them a considerable number of blood vessels, as is easily observable in figure 1, B and C.

The variation in the location of the ciliary processes is important in several ways. Iris surgery is so frequent that the anatomy at the root of the iris should be well understood. When the ciliary processes are extensively displaced on the posterior surface of the iris, iridectomy to the root of the iris would be difficult to perform and entail more risk. If done, it must traumatize the ciliary processes. It makes a great difference in the amount of hemorrhage if some ciliary processes with their blood vessels are cut. Injury to the ciliary processes may cause postoperative cyclitis. It may also be the cause of sympathetic ophthalmia. This may be the reason why some iridectomies cause much more reaction than others.

The basis for the variation of the ciliary processes is better understood by a study of the embryology of the eye.

* From the service of Dr. Robert K. Lambert and the Garfunkel Eye Laboratory at Montefiore Hospital. I am indebted to Dr. Bernard Samuels for his advice, and for his kindness in permitting the examination of many additional specimens at the New York Eye and Ear Infirmary.

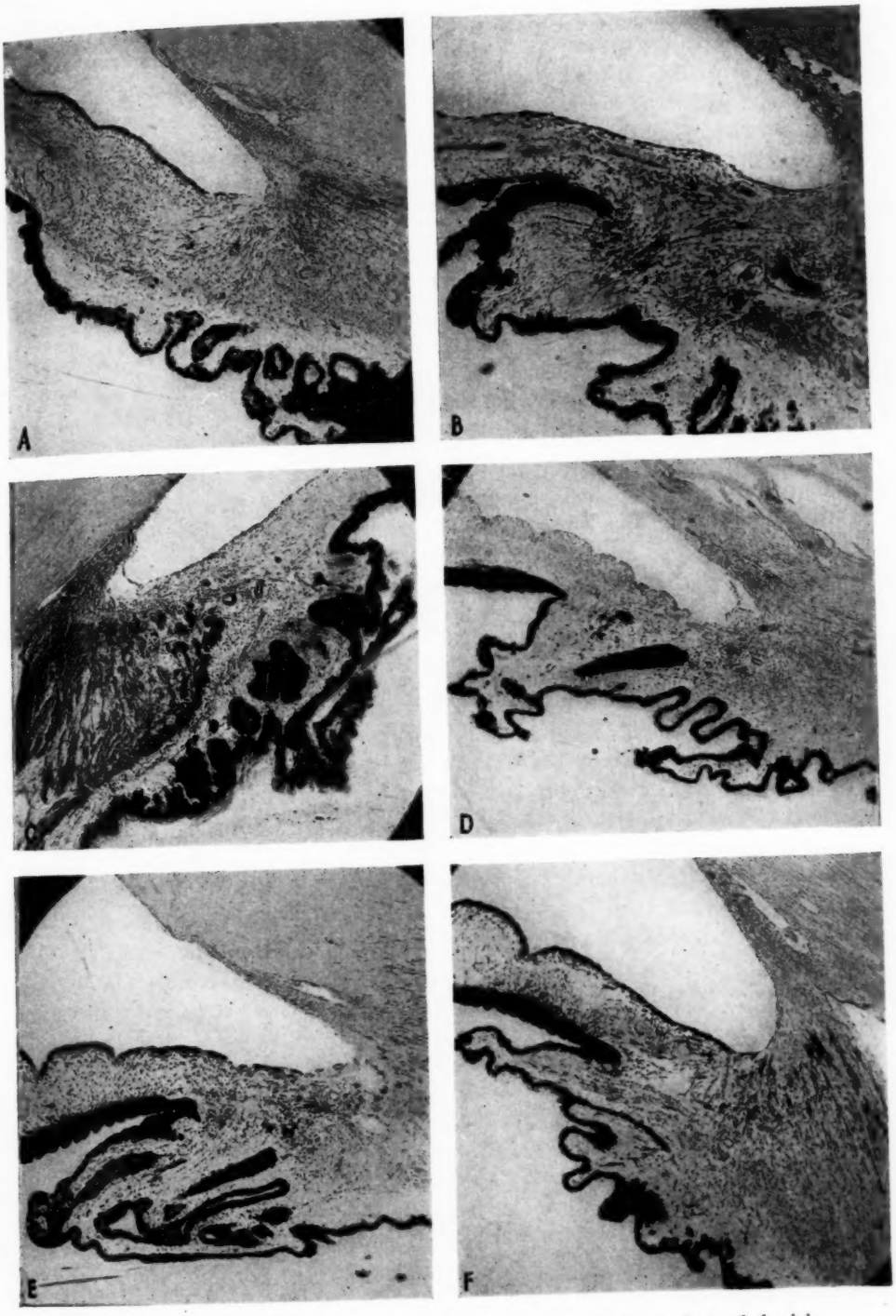

Fig. 1 (Gartner). Ciliary processes displaced to the posterior surface of the iris.

After the third month of fetal life, anlagen of the ciliary processes develop on the iris as well as on the ciliary body. At the time of birth, those on the iris usually atrophy, while those on the ciliary body develop into the mature form of ciliary processes. However, as demonstrated in another study,[8] some of the processes on the iris remain in adult life as isolated anomalous processes. Some are abridged to the regular ciliary processes. I believe that the displacement of part of the ciliary processes to the posterior surface of the iris is simply a third stage and represents a fusion of the ciliary-process anlagen that originated on the iris with those that originated on the ciliary body.

Summary

Partial displacement of ciliary processes to the posterior surface of the iris is a common variation from the normal. This is explained by the embryology of the ciliary processes. Its importance in iridectomy is discussed.

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THE PRESENT STATUS OF ANGIOSCOTOMETRY

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Whenever a new phase of medicine is developed, there is always a long interval of suspicion, trial, and reduplication of basic studies before it is safe to include, as a part of our armamentarium, the newly presented material.

In order to review the recent progress in angioscotometry, it is necessary that a word or two be said about the evolution of the basic studies. As pointed out* elsewhere,[1] "vessel stumps" had been mapped by numerous workers; but the morphologic characteristics had not been sufficiently elucidated by them to indicate any possibility of clinical application. A preliminary report was presented in 1926[2] which for the first time made evident the classical form of the angioscotoma.

During 1926,[3] 1927,[4] and 1929,[5] additions were made, not only to the morphologic basis of angioscotometry (the shape and type of distribution of the angioscotoma), but also attention was called to the phenomena of the widening effect (physiology) hitherto unrecognized.

In 1930[6] and 1931,[7] evidence was collected on which an hypothesis was suggested. This evidence included experimental data, pathologic material, and clinical studies.

Later in 1931,[8] the first suggestion of general clinical usefulness was reported with further evidence. After that date, specifically planned studies were made of certain disease entities. Thus in 1932,[9] the relation of the angioscotoma to nasal-accessory-sinus disease; in 1933,[10] to forms of edema; in 1935,[11] to glaucoma.

In 1938,[1] the material had developed so rapidly and there were so many contributions that it was necessary to review the basic principles, including the technique, and to present it in the form of a small volume.

In 1939,[12] basic studies then proceeded with the relation of the angioscotoma to oxygen deprivation; and in 1940,[13] to menstruation.

During this period, other writers had made contributions on this subject. In order to show the type and independence of their work, their studies are summarized in the following paragraphs.

Traquair[14] demonstrated the form and some of the relations of the angioscotoma in the first edition (1927) of his textbook. His studies were made on a tangent screen at two meters. In a personal communication of a later date, he remarked that he succeeded even better at one meter than he did at two meters.

Davis (1928)[15] endorsed angioscotometry as an aid to the study of intracranial lesions. In discussing this remark, Gradle stated that angioscotoma studies should help in the elucidation of the mechanism of blind-spot enlargement.

Dymschitz (1929)[16] reviewed all the basic work as it existed at that time and found it in agreement with his own work, though he used the tangent screen and otherwise modified the technique.

Dymschitz and Ilyin (1929)[17] demonstrated changes in the angioscotoma of nasal origin. This was an original contribution.

George de Schweinitz (1930)[18] listed angioscotometry as a worthwhile contribution to modern ophthalmology.

Duke-Elder (1932)[19] in the first vol-

* The work of earlier contributors is discussed under the heading of "History," in this reference.

ume of his textbook apparently misunderstood the anatomic-physiologic basis of angioscotometry. Although much of the literature was available at the time of his writing, he failed to refer to it. He labored under the mistaken idea that the actual shadows of the retinal vessels were being mapped. He contended that such extreme accuracy would be demanded that the results could not be relied upon. He suggested that the defects might arise from the Troxler phenomenon.

Jonas Friedenwald (1933)[20] writing in a work on arteriosclerosis, thought that possible artifacts might have to be eliminated from the original work. In his opinion, however, the value of the work could not be overestimated, since it represented the first attempt to use the efficiencies of the visual function as an index of the efficiency of the local circulatory mechanism.

Lambert and Weiss (1933)[21] by a well-planned study showed that pseudoneuritis and pseudoedema could be differentiated from the true forms by angioscotometry. This was an original contribution. It will be noted that their work has been verified by other workers.

Bailliart, Baldenweck, and Tille (1934)[22] used angioscotometry in following the progress of retrobulbar neuritis, when treated surgically. This was a new application of angioscotometry. They also showed a relation of retinal arterial pressure to angioscotoma changes in cases of nasal sinusitis after intranasal intervention.

Spaeth (1934)[23] demonstrated the intimate relations of angioscotoma variations to swelling of the nervehead in a case of arachnoiditis, in which peripheral-field studies showed no modification.

Magitot (1934)[24] made studies before and after carotid sympathectomy in cases of retinal and optic-nerve affections and found modifications of the angioscotoma. This was an original observation.

Dubois (1935)[25] reported an original study showing modification of the angioscotoma in a case of twin pregnancy, complicated by glaucoma.

Magitot (1935)[26] reviewed the entire subject of angioscotometry and then duplicated and corroborated the basic work.

Dubois (1935)[27] in an original study found that parenteral injection of gold salts was associated with modification of the angioscotoma.

Puglisi-Duranti (1935)[28] besides reviewing numerous aspects of the subject added the original observation that there is widening of the angioscotoma in myelogenous leukemia and in the neighborhood of minute hemorrhages. This writer also corroborated the usefulness of this method by studies in papilledema, nasal-sinus disease, and in doubtful cases of glaucoma.

Malbran (1936),[29] in the second edition of his standard textbook on the visual field, included references to the normal and pathologic angioscotomas and such literature as had appeared at the time his work was prepared.

Kalkutina (1937)[30] studied 100 cases for the size and form of the angioscotoma. The technique used was that of Dashevsky. This study agreed with those originally presented.

Dashevsky (1937)[31] duplicated studies, using his own technique, which demonstrated the usefulness of angioscotometry before and after instillation of pilocarpine in cases of glaucoma. He considered angioscotometry to be the most sensitive of all field methods for the study of such cases.

Dubois-Poulsen (1937)[32] found changes in the angioscotoma after applications to the sphenopalatine ganglion. This was an original study and called forth a discussion of Canuyt, who had made similar studies in the treatment of

retrobulbar neuritis and also during the treatment of early syphilitic optic atrophy by applications to the sphenopalatine ganglion.

Dashevsky (1938)[33] elucidated certain aspects of angioscotometry and described his own technique.

Peter (1938)[34] in his standard work on perimetry felt that he was able to endorse, from his own experience, the use of angioscotometry to disclose early evidence of circulatory pathology. He also suggested that it was helpful in making refined studies of defects of nerve-fiber origin.

By the time his second volume appeared, Duke-Elder (1938)[35] apparently agreed that the angioscotoma was not an artifact, but he still maintained that it was outside the scope of clinical usefulness. He still had an exaggerated idea of the difficulties of fixation, and did not refer to the extensive clinical evidence presented by numerous workers corroborating the original clinical studies and extending the usefulness of the method. He made but one reference to the literature more recent than the original preliminary report. In the third volume of this monumental work (1940), this author is less critical.

Evans and McFarland (1938)[12] applied the technique of angioscotometry in a study of "The effects of oxygen deprivation on the central visual field." This was an original contribution not only on the subject of angioscotometry but also to the literature on the effects of high altitudes on the human economy.

Traquair (1938),[36] in the recent edition of his textbook, gave much space to the consideration of angioscotometry and referred to the literature. At the 1940 meeting of the American Ophthalmological Society he presented a study on the relation of the fundus picture to the angioscotoma.

Magitot and Dubois-Poulsen (1939),[37] found angioscotometry to be of definite value in the diagnosis of papilledema. This study corroborated the work of Lambert and Weiss.

Thomasson (1939),[38] in reviewing the current edition of Peter's work on perimetry, stated that he did not think that the author should have included angioscotometry. Thomasson contended that it had not been accepted by the authorities. It was obvious that he was unfamiliar with the literature and progress of angioscotometry. This seemed odd, in view of the fact that Peter, Traquair, and Malbran had given elaborate references.

Rosenthal (1939)[39] found changes in the angioscotoma associated with the administration of sulfanilamide. This was an original contribution and was the first in which the narrowng phenomenon had been reported.

Magitot (1939)[40] summarized the subject of angioscotometry and pointed out its clinical applications, passing his judgment on his own experience. He stated that Traquair originated the name (angioscotom). In a personal communication however, Traquair, has stated that he did not originate this term. This is important because the present writer described the derivation and application of this word in his early contributions, as originating with himself.[3,1]

Rosenthal (1939)[41] also made original studies showing a relation of oxygen inhalation to angioscotoma changes.

Rosenthal and Seitz (1940) investigated changes in the angioscotoma associated with the oral administration of Benzedrine sulphate[42] and of strychnine.[43] Both constituted original observations.

Jones (1940)[44] read a paper before the Medical Society of the State of New York, in which he presented angioscotoma maps of subjects suffering from

undulant fever. This study was original.

Evans (1940)[13] contributed to the original studies by showing a relation of the angioscotoma to the menstrual cycle and pointing out a possible aid in the recognition of beginning pregnancy. In discussing this paper at the 1940 meeting of the American Ophthalmological Society, Berens showed similar studies.

Vishnevskii (1940)[45] reviewed the entire subject, giving special attention to glaucoma and optic neuritis. He stressed the value of comparing the scotoma of one eye with the scotoma of the other.

Dashevski (1940)[46] discussed the effect of darkness on the blind spot and of the angioscotoma of the glaucomatous eye.

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THE ORTHOPTIC TREATMENT OF ALTERNATING SQUINT*

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Whatever the special field of interest of each of us, ophthalmology or orthoptics, we have one common objective; namely, helping patients to obtain comfortable binocular vision. Orthoptics works in a limited area, but when "Alternating squint" was suggested to me as a reasonably limiting topic for a paper, I found it very difficult to arrange my ideas around such a title. Consultation and discussion with others has enabled me to understand why this is so, and as I believe my conclusions are of value to all who work in this field, I am sharing my discoveries with you as a part of this report. They are easy to summarize: I do not find myself treating cases of alternating squint, nor accommodative squint, nor phorias; I find my work consists of treating patients.

Treatment of all types of orthoptic patients consists of one invariable pattern: the establishment of a goal, analysis of the patient's status in relation to that goal, and the process of helping the patient to travel from his starting place as far along the road to his goal as it is possible for him to go.

In all cases the goal is the same: comfortable, habitual binocular vision in a variety of fields. In every case the starting point and the path taken are different, for no two individuals are alike. For this reason it is not possible to make a specific pattern of orthoptic treatment and present it as a routine method of reëducating squinting eyes. It is, above everything else, an educational process and subject to all the individual adjustments and variations called for in the techniques of education.

At various stages, however, the progress of different individuals coincides. It is possible to establish minor goals and check their achievement. In this way a broad pattern of technique is established and outlines of progress can be made. This report is limited to alternating squint.

The first step is to find out what the patient can do, to analyze his status before beginning his training. Since orthoptic treatment is always time-consuming, it is especially important that no time be wasted in makng the patient practice skills he already has. Find out promptly what he can do and start him on the next higher skill in binocular vision. The lowest step on the ladder is normal correspondence, or true binocular perception. If he has anomalous correspondence it is useless to try to develop any binocular skills, since all his subjective perceptions will be false.

Anomalous correspondence may be detected either by the afterimage test recommended by Bielschowsky, or on the major amblyoscope. The latter method is quantitative, giving the exact angle of anomaly. It is satisfactory in dealing with children as young as three years of age.

The major amblyoscope is also the best instrument for correcting anomalous correspondence—so far as I know there is nothing else that does work. It takes from 4 to 24 half-hour sessions, and occasionally more, to cure it. I have had uniformly successful results in developing true correspondence by means of the synoptophore. Of all the patients submitted to me who had anomalous corre-

* Read before the Joint Meeting of Ophthalmologists and Orthoptic Technicians in Chicago, October 20, 1941.

spondence I have corrected the condition in all but the current cases; two of these patients have had over 50 lessons and are much improved but not cured. I consider a patient cured when he has a true afterimage and when his subjective and objective measurements on the synoptophore coincide. My principal method of treatment is simultaneous stimulation of corresponding areas by very rapid motion: I lock the arms of the synoptophore at the patient's objective angle of deviation and move them as fast as necessary to get the patient to project to targets in the same direction; that is, see the lion in the cage. In this way both maculas are stimulated by a method calculated to arouse conscious perception of both images. The speed of movement is gradually decreased until none is needed, and practice in finding the "true direction" subjectively is introduced. From this stage I add fusion practice until the patient has reliably true responses to both simultaneous perception (unlike) and fusion (like) targets.

When anomalous correspondence is corrected the patient is on a par with a patient who has normal correspondence and is ready for step two on the ladder; namely, fusion.

In two and a half years' experience with about 200 patients I have had only one who failed to show fusion on the synoptophore. This patient was an alternater, a young woman aged 20 years. She had 40$^{\Delta}$ exotropia, and after orthoptic treatment failed to arouse fusion, she was operated upon with good cosmetic results and has had constant diplopia in the year since. She has not had postoperative lessons.

In trying to establish fusion I do not classify patients in any special way. I set the problem as: Under what conditions can the patient fuse? Is it in the position of exophoria, esophoria, or does a vertical deviation enter into the problem? Does the patient fuse readily when the position is adjusted, or must stimulation, such as motion or flashing, be added? Once fusion is established, the next step is to increase the variety of fusional experience and decrease the amount of stimulation necessary. This is achieved by practice under controlled conditions; that is, adapting the fusional problem to the ability of the patient, and checking his judgments so as to be sure that he is *perceiving* as well as merely looking. This has long been one of the weak points in orthoptic training; too much has been left to the judgment of the patient, and he is often, especially if very young, quite unable to make correct judgments about what he sees. If he has never experienced fusion he cannot be expected to recognize it infallibly. This was one of the disadvantages in using the stereoscope. It is important to tell children what they are seeing, and if the technician is to do this she must work under controlled conditions so that she knows reliably what the patient is seeing at a given moment.

There is nothing in my experience to show that alternaters behave any differently from other types of strabismic patients in developing fusion, nor am I able to detect any orthoptic classification based on the direction in which they deviate. Orthoptically, the problem is one of how they behave in fusion situations. It is at this stage of training that "horror fusionis" is observed. I have a patient with such a case under training now. She is improving steadily, but I do not know how far she will go. It is also at this stage that operation frequently is performed, and sometimes I do not see the patient again. This makes many of my histories of fusion incomplete.

In general I do not find that a knowledge of the preceding operative treatment affects the orthoptic treatment. It greatly

affects the status of the patient, and when operation intervenes during treatment, it changes the status suddenly, thus affecting the speed with which the patient climbs the ladder of binocular vision; but it does not affect the method of training nor the general orthoptic procedure.

When fusion is established, the next step is to analyze the possibilities of amplitude. This really means: Can the patient learn to maintain fusion under difficulties, or under what are, to him, problem conditions? In developing amplitude, he is asked to maintain fusion while his eyes move. This is frequently a stopping place in orthoptic training. For various reasons the patient may not be able to develop amplitude. Sometimes surgery corrects this difficulty, but even surgery is inadequate. Such patients remain alternaters for casual seeing. They can show fusion only as a skill or stunt under favorable conditions on the synoptophore or, perhaps, on instruments based on the principle of the stereoscope.

With the establishment of fusion with possibilities of amplitude I start the patient on the development of binocular perception in casual seeing; that is, diplopia. This means the breakdown of suppression, generally the most difficult and tedious step in orthoptic training. This perception of diplopia is the normal binocular response to binocular vision when one image falls on the fovea of one eye and the other image, due to the angle of squint, fails to fall on the fovea of the other eye. Nature solves this problem for the patient by teaching him to suppress one image. To overcome suppression he must learn that it is entirely possible for him to perceive with both eyes at once, although it gives him diplopia, a novel experience for him, and often sufficiently disagreeable to make him fight against it for a long time.

He is first taught control of alternation. Many patients alternate at random. This means they look first with one eye and then with the other, without realizing what they are doing and without the conscious direction of their attention. By learning steady monocular fixation, if necessary with a cover over the other eye to help them, then alternation at command, and proceeding to subjective choice of fixation ("I will look with my left eye"), he gets control of monocular fixation and has the necessary background for binocular fixation.

The use of a red cover over one eye is useful at this stage, especially for children. Their alteration is directed to the red image and the white image, and they are encouraged to find both at once. I carry on the process simultaneously on the instruments and on the most everyday situations I can devise: hand apparatus, picture books, toys mounted on sticks for convenience in movement to and fro, stickers on the wall, and the like. Since the alternater is by definition a suppresser, this condition invariably exists in alternating squint. The depth of suppression varies very much, and is not in proportion to the amount of squint. I had a 45$^{\Delta}$ exotropia with no fusion on his first visit get diplopia on casual seeing in eight visits. He immediately began to practice fusion, entirely overcoming his deviation and becoming a binocular reader in two months.

It may not seem strictly in the province of this paper to discuss cases of phoria in which there is alternation, but I find many such. Even patients with mild cases of convergence insufficiency often prove to be alternaters while reading and have to be taken through the steps of breaking down suppression to get comfortable binocular vision. When using the eyes for near work they show symptoms of distress that are out of proportion to their phoria measurements. I had a book-

keeper who found columns of figures jumping so badly that only the most fatiguing concentration enabled him to keep his place in the columns. Tests showed that he was a random alternater; with a red cover over one eye he saw the page first red then white then spotted. A bar-reader was single and moved on the page. By practicing he quickly learned to keep the page a blended "pink" and to get the correct diplopia on a bar-reader. Once he recognized this problem he rapidly learned binocular fixation and obtained relief from his symptoms. Several children with reading problems at school proved to be alternaters and improved their reading skills as soon as they learned to be binocular readers.

Cosmetically satisfactory postoperative patients are a very stubborn class of alternaters. Their deviation is slight and they promptly develop enough amplitude for comfortable fusion, but it is very difficult to get them to use binocular vision. Even when no diplopia will result, due to the operative correction of their deviation, and even after prolonged experience of fusion on the synoptophore, they refuse binocular vision on casual seeing. Measurements of the fusion status of such patients after operation often look as if binocular vision had been established. The doctor often depends upon the stereoscope for such measurements, and a patient is able to fuse with depth perception on an instrument who fails to practice this skill in casual seeing. Orthoptic analysis frequently shows that binocular *habits* are still remote. This is a good example of a type of patient who cannot be usefully classified by ophthalmic diagnosis nor by orthoptic technique. He has to be studied as an individual to find out the cause of his deep suppression. Patients of this kind are apt to turn out to be "problem children"; that is, psychologic problems. They are perfectly comfortable in their monocular adjustments, and it is entirely outside of their age interests to have a lot of grown-ups fussing about whether they can use bar-reader or red cover. They try to find out what are considered the "right" answers and repeat them in a parrotlike manner, or they enjoy the interest they are creating and go on doing things the wrong way. To find out what will encourage them to try to use both eyes takes a long study of the interests and responses of each patient.

The development of amplitude is coincident with the breakdown of suppression, for when the patient maintains binocular fixation on moving targets he must either fuse or suppress. As he suppresses less he fuses more, accepts the problems of amplitude. For this reason by the time suppression is entirely broken down a patient usually has a fair amount of amplitude and develops his maximum quite easily, within the limits of his motor ability. This is a second place in orthoptic training where surgical help is often indicated.

Once a patient has achieved true binocular perception with no suppression and some amplitude he is ready to transfer his skills to casual seeing. If he still has a deviation he will have diplopia, but since he has amplitude he can make the two images approach each other, and if he has enough amplitude he can make them fuse. This is the critical point in orthoptic training—the aim of all the previous work. Step by step each objective has been attained. If he can now fuse for casual seeing he can practice in everyday situations until it becomes easy. He is ready for bar-reading if he is old enough to read, and, indeed, I find it very difficult to perfect nonreading patients in binocular habits. Reading is the only every-day situation in which the eyes meet a fairly constant problem; that is, the factors of accommodation and con-

vergence are stabilized at a specific distance and the eyes are not required to make rapid shifts in adjustment. Young children whose attention changes rapidly, so that they look from picture book to narrator, out the window, and elsewhere, are demanding more adjustments of the eyes and thus are required to use the greatest binocular skill when they are least prepared for it. Once a patient can bar-read, he has both a skill and a check on his attainments. Use develops into habit, and the patient is ready to be discharged, cured.

In reviewing this outline of procedure you will note that it is an educational process throughout. Given the individual, who is the patient, you are exposing him to controlled stimuli in the effort to bring about certain desired responses. If success is to result the stimuli must be selected in such a way that they are within the limits of the ability of the patient to respond to them, and they must be presented in such a way that the patient desires to respond. It is only desire that overcomes difficulties. Too often orthoptics fails at this step. Clever targets, ingenious instruments, meticulous measurements make the situation exactly right for the patient, but he *does* nothing about it. He sits at an instrument week after week, and eventually is given up as not suited to orthoptic training. It is my earnest belief that this is because of the failure to develop *responses* from the patient. The orthoptic skill here needed is pedagogics. Successful orthoptists are primarily good teachers. Many, fortunately, are naturally clever at teaching, but teaching skills can be learned, and orthoptics will make progress very fast when the patient-orthoptist situation is analyzed in the light of modern teaching techniques.

Alternaters have established a behavior pattern in the use of their eyes. We wish them to use a different pattern, binocular vision. That is our aim. Analyze the situation: What can he do? Establish an objective. Let him experience it. Teach him to want it. Show him how to get it. Advance to new objectives. Give him standards by which to recognize his gains. How does he use his eyes? What does he see? Constant subjective practice is important. Our objective measurements are of no use to the patient. They are for our guidance in establishing procedures, never an end in themselves. We are working to change the behavior habits of an individual. This is an educative procedure. It involves behavior, emotions, drives, and above all stimulus and response; finding the right stimulus to arouse the desired response.

In conclusion I should like to review the stages at which orthoptic training proves a failure in establishing binocular vision. It is not in normal correspondence. It is rarely in fusion. It is frequently in developing amplitude; that is where surgery is necessary. It is also frequently at the point where performance transfers to habit. Every patient who comes as far as that and fails is a failure in orthoptic skill, and it is my contention and the thesis I should like to leave with you, that such failure is due to ignorance or lack of skill in sound educational procedure.

490 Post Street.

NOTES, CASES, INSTRUMENTS

TO MEASURE THE PUPIL

EDWARD JACKSON, M.D.
Denver, Colorado

The kind of pupillometer described in the December, 1941, number of the Journal, by Dr. David G. Cogan (p. 1431), is worthy of more attention than it has heretofore received. It was first described in the Philadelphia Polyclinic in 1887, volume 4, page 362, in a still simpler form. It cannot be made in a factory to sell at a profit. So it has not been widely advertised; and many who might use it with advantage have never heard of it. As Dr. Cogan says, it can be made out of "a piece of cardboard with a pin and a millimeter rule." It is not necessary to have holes for each half millimeter; one-millimeter intervals are better.

The patient makes the observation. It must be explained to him what he will see: one hole, two holes overlapping, two holes just touching each other, or two holes separated. The measurement is made with the two holes just touching each other, and can be repeated until the doctor feels sure the patient understands and is giving correct answers.

With such a card the consensual reaction of the pupil can be studied by holding the card before one eye and excluding or admitting light to the other alternately. Placing the pupillometer before first one eye and then the other, and alternating this test, one may detect an early loss of reaction in one eye. This is a significant symptom in some diseases of the central nervous system.

The cardboard should be black, in order fully to exclude light from the eye before which it is placed. It should be thin to avoid the tube effect of a hole through thick cardboard. The holes should be punched with a fine needle, thrust in to make the holes all of the same diameter. That is, thrust in to where the needle becomes of uniform diameter. The holes may be placed by drawing two lines from the same point at one end, and separated 10 millimeters at the other end. Then, across these, draw 10 parallel lines at intervals of 10 millimeters. These lines must be placed at right angles to a line halfway between the first two diverging lines. The pairs of holes, punched at the crossings of parallel and diverging lines, will then vary one millimeter in the space between them. It is worth while to make such a card, to understand the principle that it embodies. Then the doctor should use it on his own eyes, under different conditions of lighting, at different times of the day, and after different kinds of preëxposure to light. After the technique has been mastered it can be used with advantage and satisfaction on patients. Not many patients need to have their pupillary reactions measured and recorded. But for the few who do need such an examination, such a card cannot be replaced by any of the more elaborate and expensive instruments.

Republic Building.

CONGENITAL CYST OF THE SCLERA

JOSEPH F. DUANE, M.D.
Peoria, Illinois

Scleral cysts are uncommon. Most of them are epithelial inclusions secondary to trauma. About 30 cases of traumatic origin have been reported. Nontraumatic congenital scleral cysts are rare. One such case is here reported:

J. O'N., whose birth was premature (at seven months), was 18 months old

when first seen on March 18, 1941. Except for his left eye, there were no congenital defects. At birth a blue spot, the size of a pinhead, was noticed by the mother "in the white" of this eye on the nasal side and near the cornea. This spot increased in size for about three months and protruded. Then it remained stationary for about nine months. When the child was about one year old he had an upper respiratory infection, and the mother thought the lesion had gradually increased in size from then on. When examined again, on March 31, 1941, the growth appeared to be about the same size as on the first examination. Both examinations were made on a struggling, unanesthetized child. A diagnosis of conjunctival cyst was made and operation advised.

Fig. 1 (Duane). Schematic drawings, made by Dr. C. D. Sneller, showing the various stages in the development of the scleral cyst. A, subconjunctival portion; B, scleral portion of cyst.

At the time of the operation, on April 10, 1941, a careful examination of the left eye under ether anesthesia could be made. On the nasal side of the cornea, adjacent to, and concentric with, the limbus there was a grayish protrusion extending from about the 9-o'clock position downward to the 6-o'clock position. It extended nasally about 6 mm. at the top and narrowed to about 4 mm. below, was about 9 mm. in its vertical diameter, and protruded about 4 mm. Its limbal edge was sharply perpendicular, the nasal border more sloping. The growth was covered by a slightly cystic avascular conjunctiva, which could be moved freely over the protrusion. Tension of the eye to the fingers seemed normal. The protrusion fluctuated on palpation, and pressure on it made no visible change in its relation to the anterior chamber (fig. 1).

The conjunctiva was dissected free from the limbal edge and retracted. There was no line of demarcation between the bulbar sclera and the protruding sclera. The protrusion was punctured at its upper border by a fine knife-point. A clear fluid squirted out, and the protrusion flattened down. There was no loss of fluid from, nor any change of relation in, the anterior chamber, nor was there any perceptible change in the pressure of the eye to the fingers. By palpation with a hook through the flattened scleral tent a resisting inner wall could be felt. A piece of the collapsed sclera was removed at the site of the puncture, and it could then be seen that the growth was a cyst, with about one half of the thickness of the sclera making up the outer wall and the other half the base. The remainder of the outer wall was then removed, leaving a shallow scleral depression, through which the blue color of the uvea could be seen. The base of the cyst was scraped gently with a knife edge to remove, if possible, any secreting cells. The limbal dissection of the conjunctiva was extended to the 12-o'clock position above and to the 4-o'clock position temporally. Thick, wide, H-shaped flaps were fashioned to overlap each other and cover about one-fifth of the cornea. These were held in place by two mattressed silk sutures above and

two below, and a running silk suture was used to close the conjunctival defect on the nasal side.

The outer wall of the cyst was removed piecemeal and lost so that no histologic examination of tissue could be made.

The eye healed uneventfully. The stitches were removed under an anesthetic after 12 days. The tension of the two eyes to the fingers seemed equal and normal. At that time and afterward the flaps appeared to be holding firmly. Eight months after the operation, there was no evidence of protrusion, the cornea was clear, and the eye was not injected (fig. 2).

Some time after the child was discharged from the hospital, I learned that this patient had been previously seen by a colleague here, Dr. C. D. Sneller, who had made the correct diagnosis of scleral cyst. He had also made schematic drawings of various stages in the development of this cyst, a photograph of which he graciously offered to me. The photograph is used in this report and is hereby gratefully acknowledged.

Search through the literature for information concerning scleral cysts revealed very little. In most works on ophthalmology there was nothing except the scant statement that "cysts of the sclera may occur." Duke-Elder,[1] however, in a short article on congenital cysts of the sclera, reported seven cases. In his opinion the etiology may not always be the same.

Two additional cases were found to have been reported by Wernicke[2] and by Seefelder.[3] A few cases (about 10) of congenital corneo-scleral cysts are to be found in the literature. The evidence in these reports seems to show that they developed from primary scleral cysts by dissecting into the layers of the cornea. Hence the indication for early operation while the cyst is still limited to the sclera. In one case the cyst was simply punctured and it promptly refilled. The youngest previously reported age at which an operation for a congenital scleral cyst had been performed was three and a half years. This was done by Custodis upon a patient whom he had first seen when the child was between four and five months old.

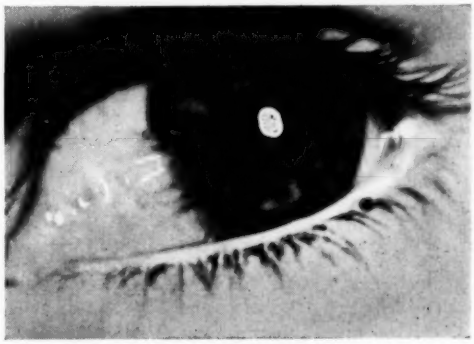

Fig. 2 (Duane). The eye eight months after operation.

The last report of a case of congenital corneo-scleral cyst was made by Vrolijk;[4] in it he reviews the various theories of etiology and correlates them to the investigations of Sondermann[5] on the development of the canal of Schlemm. Vrolijk concludes that the constant location of congenital scleral cysts at the limbus is best explained by presuming that their origin is from pinched off vessels which missed connection with the canal of Schlemm in their embryologic growth.

418 Jefferson Building.

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SOCIETY PROCEEDINGS

EDITED BY DR. RALPH H. MILLER

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

February 3, 1941

DR. MORRIS DAVIDSON, *presiding*

GRAPHIC REPRESENTATION OF BINOCULAR MEASUREMENTS

DR. EMANUEL KRIMSKY discussed this subject and its clinical applications during the Instructional Hour.

UNILATERAL MICROPHTHALMOS ASSOCIATED WITH CONGENITAL ANTERIOR IRIDIC MEMBRANE, GLASS MEMBRANE, AND SYNDACTYLIA

DR. MILTON L. BERLINER reported two cases of left unilateral microphthalmos occurring in female first cousins, aged 5½ and 1½ years. Both showed anterior synechiae extending from the lesser circle of the iris to the posterior cornea, the site of the corneal attachment being marked by the presence of a hyaline membrane. In addition, there was partial syndactylia of the fingers and toes in both cases. This syndrome differs in some details from any previously reported.

Dr. Berliner reviewed the theories of the pathogenesis of microphthalmos and the embryogenesis of defects of the extremities. He believes that such localized congenital malformations result from an arrest or deviation of development occurring in embryonic rudimentary structures due to some unknown defect in the germ plasm.

MARFAN'S SYNDROME

DR. ANDREW RADOS presented 10 cases of lens dislocation, 7 of which showed typical arachnodactyly; 6 were bilateral and 1 unilateral. In these cases and also in over 200 others reported in the literature, frequent symptoms are elongation of the long bones, skeletal changes, anomalies of the heart and those of the ears. Heredity may be a factor in this syndrome, but in the majority of cases no hereditary tendency can be found. This syndrome is probably genetic in origin and may be explained on the basis of a by-mutation of the genes. The endocrine etiology is not acceptable because these malformations are present at birth, and this is not true of hormonal disturbances.

CAROTID ANEURYSM ASSOCIATED WITH OPTIC COLOBOMA AND OPHTHALMOPLEGIA

DR. LESTER STEIN reported the case of a negro woman, aged 55 years, who had a sudden onset of left cephalalgia, followed shortly by total left internal and external ophthalmoplegia. General examination disclosed marked hypertension and arteriosclerotic heart disease. Ophthalmoscopic examination showed hypertensive retinopathy, coloboma of the optic nerve of the left eye, and choroiditis. X-ray studies of the skull showed erosion of the clivus, the sella turcica, and of the greater wing of the sphenoid. Cerebral arteriography with Diodrast disclosed a round aneurysm of the internal carotid artery adjacent to the sella as it passed through the cavernous sinus. At no time was there noted any thrill, murmur, or bruit, either subjectively or objectively. The patient's condition was followed for over a year and showed improvement under symptomatic treatment.

OCULAR TRICHINOSIS

DR. CHARLES LITTWIN presented a case in which the patient complained of severe frontal headache, bilateral lid swelling, conjunctival chemosis, and slight papilledema—all occurring within two days. A tentative diagnosis of cavernous-

sinus thrombosis was changed to trichinosis when the blood count showed 28 percent eosinophiles. This was confirmed by finding *Trichina spiralis* in a biopsy from the left deltoid muscle. At no time did the patient complain of muscle or joint pains, nor was there any history of gastroenteric disturbance. The patient was treated with sulfanilamide and blood transfusions and was discharged on the twentieth day.

Congenital night blindness

Dr. Adolph Posner reported three cases of congenital night blindness in children. The parents had normal night vision in every case. All three were highly myopic and none showed rod function when tested for dark adaptation. There was no improvement under vitamin-A therapy.

Discussion. Dr. Joseph Mandelbaum stated that these three cases showed dark-adaptation curves of practically normal cone function with no demonstrable rod function. The value of plotting cone and rod function separately, as is possible with the use of the Hecht-Schlaer Adaptometer, is clearly shown here. Advanced disease affecting both rod and cone function—which might give similar final light thresholds—may thus be readily distinguished and separated.

In the differential diagnosis of these cases, vitamin-A deficiency and retinitis pigmentosa sine pigmento must be considered. The former can be eliminated by the normal dietary history and the failure of reaction to vitamin-A therapy; moreover, a deficiency severe enough to eliminate rod function completely would have affected cone function more markedly. Ophthalmoscopic examination failed to reveal any signs pointing to an early retinitis pigmentosa. In the absence of severe systemic metabolic disturbances, the remaining diagnosis is hereditary nyctalopia with absence of rod function.

Retained wooden intraocular foreign body

Dr. Sidney A. Fox reported the case of a girl, aged nine years, the cornea of whose right eye had been penetrated by a wooden splinter 3½ years ago. The foreign body left several tiny splinters between the lips of the corneal wound and lodged in the iris. There was a prolapse of the iris which retracted spontaneously, also leaving the iris ruff in the corneal wound, from the 3- to the 6-o'clock position. There was no other damage to the eye.

At present the eye was white and quiet, with a vision of 20/20. Most of the pigment left in the cornea was absorbed, and the iris splinter had become encapsulated and pigmented. Except for iris atrophy in the 3- to 6-o'clock position, the eye was quite normal. A study of the literature for the past 25 years disclosed few reports of retained intraocular foreign bodies of wood, and these all resulted in permanent visual loss of varying degree. Wood is a substance especially to be feared inside the eye.

Bilateral central-retinal-vein thrombosis

Dr. Sigmund A. Agatston reported a case of central-retinal-vein thrombosis of the left eye in a hypertensive patient. The fundus picture in the right eye was fairly normal at the time. Three years later there was a venous thrombosis in the right eye. The latter developed a secondary glaucoma and had to be enucleated, following which the left eye showed a marked functional improvement of vision. After a study of the clinical and laboratory findings, Dr. Agatston concluded that nonhypertensive sclerosis came first, essential hypertension was incidental. Thrombosis of the sclerotic retinal veins was the basis of severe hemorrhagic manifestations. The advanced

changes in the arteries seen later were not caused by the general hypertension but were secondary to the venous thrombosis. Acute glaucoma was secondary to the venous thrombosis.

Discussion. Dr. Martin Cohen asked how Dr. Agatston would distinguish between essential and malignant hypertension by a fundus examination. He did not think it was possible.

Dr. Morris Davidson asked how Dr. Agatston explained the cause of the secondary glaucoma.

Dr. Agatston in conclusion stated that in malignant cases the retinal arteries were much more attenuated and less visible than in essential hypertension. He thought that the secondary glaucoma following central-retinal-vein thrombosis was due to the blocking of the return circulation, thus causing an increase in the intraocular fluids.

BILATERAL OPTIC ATROPHY DUE TO METHYL-ALCOHOL POISONING COMPLICATED BY TOBACCO-ETHYL-ALCOHOL AMBLYOPIA

DR. BENJAMIN FRIEDMAN reported this case and showed field studies differentiating the two conditions in the same patient.

Discussion. Dr. Dewey Katz asked how it was possible to reconcile the peripheral constriction of the fields shown with the fact that most of the changes ought to be confined to the central fields in toxic amblyopia.

Dr. Friedman stated that the constriction of the peripheral fields was due to the original poisoning and that this is quite consistent with the evident optic atrophy. During the acute stages of methyl-alcohol poisoning the patient had large positive central scotomata readily verified by Weekers's test. The recent episode of tobacco-ethyl-alcohol toxemia was manifested by a caeco-central scotoma associated with diminution of vision. The vision improved with treatment, and the central portion of the scotoma disappeared within a week.

Sidney A. Fox,
Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

February 20, 1941

DR. FRANCIS HEED ADLER, *chairman*

REMOVAL OF NONMAGNETIC FOREIGN BODY

DR. EDMUND B. SPAETH presented a case illustrating the removal of a nonmagnetic copper-alloy foreign body wherein the exact choroidal localization was obtained by anteroposterior roentgenograms and wherein silver rings had been sutured about the limbus and about the approximate site of the foreign body, scleral projection. With the measurements obtained thereby, a hinged-flap sclerotomy anterior to the equator was performed at the plotted site, and the embedded foreign body removed from the choroid. Endoscopic removal and biplane fluoroscopy removal were both out of the question because the foreign body was covered over and hidden by an area of exudative chorioretinitis. Postoperative recovery was uneventful. Central visual acuity was 6/6 with correction.

Discussion. Dr. Warren S. Reese asked if the injection of the air beneath Tenon's capsule was very difficult, and if the resulting X-ray shadow was definite and satisfactory.

Dr. Spaeth, in closing, stated that air injection into Tenon's capsule was difficult, and that it did show very definitely that the foreign body was not external.

CLINICAL OBSERVATIONS ON THE TREATMENT OF SQUINT

DR. JACOB B. FELDMAN presented a paper on this subject.

RETROBULBAR INJECTION OF ALCOHOL IN CASES OF ACUTE GLAUCOMA, EXHIBITION OF CASES

DR. C. E. G. SHANNON reported five cases of painful absolute glaucoma treated with retrobulbar injection of alcohol.

He stated that Grüter was the first to advocate its use for this condition. His method, briefly, was to inject 2 c.c. of 2-percent novocaine into the apex of the orbit between the external and inferior rectus muscles, followed in five minutes by 1 c.c. of 80-percent alcohol. At the end of 15 minutes the eyeball was entirely anesthetic. All the muscles became paralyzed, and the eye was rendered immobile. There is but slight discomfort following the injection, provided the procedure is carefully carried out.

Case 1. J. S., a man aged 47 years, suffered serious injury to his right eye in 1919, and the lens became hazy and dislocated. It was subsequently removed by Dr. Hansell. Glaucoma developed one year later, 1921, and vision was lost completely in a few years. The eye, however, did not become painful until 1939. Alcohol injection was advised and accepted. Seven days later, a second injection was given and the pain was entirely relieved. There was no return of the pain in the subsequent two years.

Case 2. L. S., a white woman aged 62 years, first suffered from iritis in her left eye, and later secondary glaucoma developed. The eye finally became blind and painful. Alcohol injection was advised and accepted. She experienced very little distress following the operation and has subsequently been quite comfortable.

Case 3. A. P., a woman aged 51 years, was first seen in February, 1936. She had a chronic simple glaucoma of the right eye. The left eye was blind, with tension of 70 mm. Hg (Schiötz). A Lagrange operation on the right eye was successful. The left eye became painful two years later, and a retrobulbar injection of alcohol was followed by complete relief of pain, which has not recurred.

Case 4. A. M., a woman aged 60 years, was first seen by Dr. Shannon in May, 1940. She was suffering from absolute glaucoma, with tension of 80 mm. Hg (Schiötz) in the right eye, and chronic simple glaucoma in the left. Ten months later, the right eye became painful, and injection of alcohol was advised and accepted. She has had but slight discomfort since.

Case 5. P. W., a man aged 57 years, was admitted to Jefferson Eye Clinic on January 26, 1941, complaining of extreme pain in his left eye. The tension was 50 mm. Hg (Schiötz), and the vision equalled 6/200. The vision decreased to bare light perception and finally was lost altogether. Alcohol injection was given on January 29, 1941. Pain was temporarily relieved. A week later, pain returned and a second injection was given, since when he has had no trouble.

Grüter (1941) reported 56 cases which he had followed for 5 years, with 6 failures. Jaench (1925) reported 29 cases, all successful. Scheffels (1927) reported 3 cases, no failures. Salvati (1927) reported 3 cases, no failures. Aléxiadès (1928) reported 15 cases, no failures. Fejer (1932) reported 5 cases; there was one failure due to intraocular growth, discovered only after the injection and removal of eyeball. Malkin (1934) reported 26 cases, 4 failures.

The contraindications to this method of treatment are as follows: (1) Useful vision in the affected eye; (2) intraocular

growth; (3) sympathetic ophthalmia.

With results generally favorable and the technique comparatively simple, it would seem that this method is far preferable to enucleation in obtaining permanent relief from pain in a blinded eye.

Discussion. Dr. Sidney Olsho asked if the tactile sense was at all interfered with following the injections.

Dr. Shannon replied that no loss of tactile sense had been reported, nor had it developed in his cases.

Dr. Warren S. Reese asked Dr. Shannon whether cases showing blebs or bullae of the cornea showed improvement.

Dr. Shannon in reply stated that the bleb to which Dr. Reese referred in case 5 was an intercalary staphyloma and he believed its development was purely coincidental. It had shown little change during the preceding two weeks. He added that there were no complications in these cases whatsoever. The only complaint the patients had was occasional neuralgic pain in the temporal region and not in the eyeball itself.

Dr. Francis H. Adler asked if Dr. Shannon thought there was any possible danger of getting some of the alcohol in a nonseptic thrombosis.

Dr. Shannon said he presumed there was some danger of that. As far as he was able to discover in the records, no such accident had occurred.

Sulfathiazole in the treatment of gonorrheal eye disease

Dr. Carroll R. Mullen reported that five cases of gonorrheal disease of the eye had been successfully treated with the use of sulfathiazole internally and externally, at the Philadelphia General Hospital. The treatment with this drug had been neither more nor less efficacious than when sulfanilamide was used, if the technique of treatment and dosage was essentially the same. Treatment consisted in the use of sulfathiazole internally in a dosage of 1 gr. per pound of body weight daily for each adult or child up to 100 grs. daily. In older children and adults, the dosage was graduated according to clinical needs. A 0.2-percent solution of sulfathiazole was used locally in the involved eye by instillations every 10 minutes without interruption for the first 24 hours. Iced compresses, a solution of atropine sulphate, and a Bullar shield, when indicated, were also employed.

Experience had pointed out the necessity of nurses working in pairs on eight-hour shifts in order to accomplish the demands of this treatment. In the second and third 24 hours, the treatment was tapered off. No unfavorable blood pictures were reported in any case, nor were any toxic changes noted. The longest period of treatment extended over parts of three days before the first of three successive negative smears was obtained. The average case showed negative results at the end of the first 24 to 48 hours of treatment. No corneal complications were observed, and each patient was discharged as cured.

Discussion. Dr. Alfred Cowan said that since the institution of this method of treatment, gonorrheal ophthalmia seemed to have become one of the "least to be feared" eye conditions with which ophthalmologists have to contend. He said it was remarkable to see how much improvement was obtained in the first 24 hours, even in the most severe cases. No one who has had experience in the management of gonorrheal ophthalmia and who has not seen this method of treatment carried out would believe that the baby or adult with gonorrheal ophthalmia had ever had such an infection after two or three days of treatment. Gonorrheal ophthalmia, today, with this method of treatment can be considered an ordi-

nary eye condition and probably responds more quickly than would an ordinary case of acute catarrhal conjunctivitis.

Dr. L. F. Appleman asked whether the use of sulfathiazole internally alone would give equally good results aside from the use of the drug locally as an instillation. He believed it would be very interesting to determine whether the local applications were really necessary. In view of the fact that the drug seemed to be specific in the destruction of the gonococcus, the internal use alone might be sufficient. He had been asked a similar question during recent lectures at the Graduate School. His own experience had been so limited that he could not give a very definite answer in regard to it. He asked for an expression of opinion from those having more experience with its use.

Dr. Francis H. Adler reported some observations that Dr. Souders and Dr. Scheie had made in his department. They had found that sulfapyridine and sulfanilamide when injected into the blood stream came through into the primary aqueous very rapidly and in concentrations which were equivalent in a short period of time with that in the blood. Sulfathiazole, however, did not come through into the primary aqueous. It was found in the plasmoid aqueous after tapping the anterior chamber, but not in the primary aqueous. If one were dealing with an intraocular infection, therefore, he assumed that sulfapyridin and sulfanilamide should be used, but not sulfathiazole, unless it were used locally, as has been done in the cases reported.

Dr. Mullen, in conclusion, said that from personal observation he believed the use of either sulfanilamide or sulfathiazole in solution could be abandoned and equally good results obtained with frequent local cold irrigations. However, he did not feel justified in taking this chance because of the very satisfactory results that had been obtained with the use of the drugs in solution and internally.

In the early clinical trials with sulfanilamide at the Philadelphia General Hospital by Dr. T. A. O'Brien about three and one-half years previously, sulfanilamide internally was used in about the same doses as had recently been used with sulfanilamide drug and sulfathiazole. The irrigations, however, were much less frequent and it was only when the irrigations were increased that the remarkable results began to become evident. The immediate results, with the internal use of sulfanilamide as the chief item of therapy, were not particularly satisfactory in gonorrheal eye disease. The best results were obtained when irrigations were continued at 10-minute intervals for a full 24 hours without permitting the patient to eat or sleep without the regular interruptions for irrigations.

He said one must always keep in mind, in this treatment, that gonorrheal disease of the eye is a local condition, unless perforation with invasion into the anterior chamber occurs. There are many reports in the literature about the rapidity of appearance of this drug in both the aqueous and vitreous after either injection or ingestion. His personal opinion was that the internal use of these drugs had very little to do with the good results obtained in the treatment of gonorrheal eye disease as it was administered at the Philadelphia General Hospital. He favored the frequent irrigations during the first 24 hours and believed that iced or cold irrigations were more efficacious than those which were warm.

The use of a solution of sulfanilamide locally before operations had been practiced for some time. For more than three years he had used it as a routine preoperative care. He was unable immediately to recall the occurrence of any postoperative infection from its use. He

agreed with Dr. Cowan that gonorrheal eye disease should no longer be treated with the anxiety that was formerly associated with it.

He stressed the importance of nursing care. He had seen cases treated by the same technique in other hospitals, but had not seen any with the good results recorded in Blockley. The end results of these cases in other hospitals were quite satisfactory, but the period of treatment was greatly lengthened, and he believed this was due to the fact that only one nurse was assigned to the case. He said there was really too much work for one nurse to carry out the complete assignment. When one considered that irrigations must be given every 10 minutes, the child must be restrained, iced compresses must be applied, the child must be fed, bathed, and cared for in other respects, it was quite difficult to understand how one girl could do all efficiently.

At the Philadelphia General Hospital two nurses worked in relays under an experienced head nurse, who was well acquainted with the prescribed technique. The results were nothing short of spectacular. He wished to lay a great deal of emphasis and stress on nursing care, the irrigations which must not be given "about" every 10 minutes, but definitely *every* 10 minutes, and the importance of recording this procedure.

Warren S. Reese, M.D.,
Clerk.

SAINT LOUIS OPHTHALMIC SOCIETY

February 28, 1941

Dr. William M. James, *president*

The specific biologic therapy of staphylococcal infections of the eye

Dr. R. H. Boots presented a paper on this subject, which has appeared in this Journal (April, 1942), in collaboration with Drs. L. A. Julianelle and G. H. Harrison.

Discussion. Dr. L. A. Julianelle said that Staphylococcus is capable of reacting in infection by any of three manifestations. While most frequently involved in localized infections (that is, in abscesses of various forms), it may be highly invasive, causing a fatal septicemia. In addition, the organism may elaborate a toxin that has been shown to affect different tissues even with the induction of death. As a third property, the organism is capable of causing tissue destruction, which means the liberation of unneutralizable, toxic, split products to be followed by replacement with scar tissue. Consequently, the treatment of staphylococcal infection is not a simple matter, but requires variations according to the form of the infection, and demands surgical and various supportive methods in the more severe diseases. In exogenous staphylococcal disease of the eye, however, the infections are almost entirely mild and noninvasive. In the localized abscess, as in styes, the treatment becomes one of evacuation of the abscess with bacteriologic cleanliness, in order to prevent spread or recurrence.

Generalized, diffuse conjunctivitis without localization presents a different clinical problem. Diffuse punctate keratitis suggests the action of a toxin on the epithelial cells of the cornea, and consequently it would seem *a priori* that immunization with toxin or toxoid should raise the blood level of antitoxin and, therefore, minimize if not eliminate the effects of the infection. The influence of autogenous vaccines on infections of the eye (particularly styes and chalazia) in the past has been doubtful, since (1) such infections are self-limited, healing spontaneously in variable periods of time, and (2) vaccine therapy is usually so prolonged as to leave a reasonable doubt as

to its efficacy. Such immunization, of course, being antibacterial, influences in no way the antitoxic titer. It was on this basis, therefore, that the present study was undertaken. As has been demonstrated, the immunization with toxoid increased the antitoxic level in every one of the patients observed, yet the clinical course of the infections was not altered nor interrupted.

On retrospect, certain considerations suggest themselves in explanation of the apparent failure. Physical conditions in the conjunctival sac—moisture, temperature, nutrition—are all ideal for the proliferation of bacteria. When the normal antibacterial barrier breaks down, the organisms take advantage of these conditions to cause infection, and the less acute the infection the less defensive is the response on the part of the conjunctiva, with the result that a chronic, low-grade process occurs. Thus the setup becomes ideal for establishing a balance between bacterium and conjunctiva. In the case of Staphylococcus, the organism is not invasive, since the infection remains localized, and the toxin it produces is either impotent or unabsorbed, since staphylococcal conjunctivitis induces no measurable generalized reaction. Consequently, the infection becomes a purely surface phenomenon, penetrating not even the epithelial cells. What immunity is elaborated by the tissues in such cases is highly localized (that is, in the epithelial cells), and with constant desquamation going on, the immune cells are constantly eliminated, so that the degree of immunity never reaches a high point. It may therefore be that immunization with toxoid, as accomplished in this study, is insufficient, since the antitoxin never reaches the actual site of the infection.

Dr. William Meinberg said he had been in the habit of taking smears and scrapings in both acute and chronic cases of conjunctivitis. He wished to ask Dr. Julianelle if there is any value in the taking of routine smears and scrapings in these cases. Only rarely has he been able to demonstrate organisms under the oil-immersion lens.

Dr. A. D. Calhoun asked whether any of these patients had a recurrence of styes or did just an occasional stye appear. Did any of them have as many as 15 or 20 styes?

Dr. T. Sanders asked whether there was any evidence that conjunctivitis is not due to the toxins of staphylococci, but due to hypersensitivity to the organism itself.

Dr. Carl Beisbarth thought that Dr. Julianelle belittled staphylococci somewhat. Last year he had had to deal with a severe wound infection after cataract extraction. He used two bottles of sulfathiazole tablets along with everything else. Could the use of toxoid be combined with sulfathiazole?

Dr. B. Y. Alvis said that he had enjoyed this paper and admired its thoroughness, but was not certain whether Dr. Julianelle should be classed as a benefactor or a destroyer of ideas. No sooner do we think something is very good, than he proves it to be scientifically all wrong. We had a patient, who came in preparatory to cataract extraction, who had a severe acne rosacea. He was treated with the toxoid and local treatment recommended by Dr. Julianelle. The conjunctiva became clear of staphylococci, the rosacea became clear, and following the cataract extraction no infection occurred. We also had two or three cases of recurring styes in which the styes stopped with the use of toxoid.

Dr. Julianelle, in closing, said in answer to Dr. Calhoun that when a patient has had 32 successive styes, it would seem that the time had about come for a spontaneous termination, regardless of the treatment employed.

In answer to Dr. Meinberg he said that

in chronic, low-grade infections, such as those discussed this evening, scrapings or smear preparations are of little help in diagnosis. The organisms are not numerous and, therefore, are missed in such preparations. It is necessary to employ cultural methods, particularly if later a distinction is to be made between nonpathogenic and potentially pathogenic staphylococci.

In answer to Dr. Beisbarth, Dr. Julianelle said that he had not had the opportunity of treating staphylococcal conjunctivitis with sulfathiazole and knew of no literature on the subject. This drug has been used with a good deal of success in treating generalized staphylococcal infections at both the Mayo Clinic and the University of Minnesota, a success far superior to the records obtained at the Children's and Barnes Hospitals in Saint Louis. It might be interesting to apply the drug directly in the conjunctival sac.

In answer to Dr. Sanders he said he did not know that he could answer the question whether in the patients studied the conjunctivitis was due to an allergy to staphylococci or to an infection by these organisms. He thought the process is primarily an infection rather than a hypersensitivity.

Dr. R. H. Boots, in closing, said that as far as styes are concerned, he personally had as much faith in Dr. Meyer Wiener's treatment as in any other—10-percent sulphuric acid over a period of 10 days. This treatment has been used in the Clinic with good results. In his opinion styes are a self-limited infection, and he thought that the bacterial cleanliness of which Dr. Julianelle spoke should be observed.

Intravenous administration of sodium nitrite in choroiditis

Dr. Allen Calhoun presented a communication on this subject.

Discussion. Dr. John Green said that one hesitates to comment on a single case, especially as that case was not one of exudative choroiditis. His patient appeared with signs of uveitis associated with a central corneal lesion resembling disciform keratitis. There were cells in the deep anterior chamber, keratitic precipitates, contracted pupil, and increased tension. The patient had undergone a good medical examination which revealed nothing of significance. He admitted having had a venereal sore 15 years ago. The blood Wassermann test was negative. He had been under treatment for prostatitis, which had greatly improved. His tonsils had been removed. There was evidence of pus in the middle meatus. The ethmoid cells were exenterated.

Suprarenin bitartrate failed to dilate the pupil and had no effect on the tension. With no improvement after two weeks, Dr. Green began treatment with sodium nitrite given intravenously and continued daily until the patient had 15 doses. One-percent atropine was now used. He began to show improvement after the third injection; by the fifth injection the pupil had dilated widely and the tension began to come down. Six weeks after beginning treatment the central corneal haze was still present but was not so dense. Keratitic precipitates had almost disappeared; the pupil was wide and the eye white.

Dr. C. Beisbarth said that shortly after the publication of Duggan's paper he had had a case of acute choroiditis. He could not get sodium nitrite ampules and, while waiting for it, used erythrol tetra nitrate. There was some improvement after the use of sodium nitrite. He did not know whether the oral administration of the drug had been of benefit.

Dr. A. D. Calhoun, in closing, said that the preparation of sodium nitrite was

made by the Metropolitan Drug Company of Saint Louis. They used the crystals of sodium nitrite in distilled water. Dr. Beisbarth's experience of using the oral administration prior to the intravenous injections probably had an effect just as good except that the results of sodium nitrite are supposed to las. a little longer. The Mayo Clinic used sodium nitrite, 0.5 grain every 20 minutes, for 6 doses and took the blood pressure at 1½ hours, 3 hours, and every hour for 6 hours after the drug was given by mouth; and they reported lowered tension.

A CASE OF UVEITIS DUE TO TAENIA SAGINATA

DR. CARL BEISBARTH presented a case report on this subject.

Adolph C. Lange,
Editor.

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THE SPRING MEETINGS

The seventy-eighth annual meeting of the American Ophthalmological Society was held in Hot Springs, Virginia, on June 1st, 2d, and 3d. Eighty-seven members and 27 guests attended. Allen Greenwood presided and opened the meeting with a few pleasing reminiscences of early days of ophthalmology in the United States. The program that followed consisted of the usual pattern of clinical and research papers, the former predominating. On the whole it exceeded expectations, and several outstanding contributions were made, notably that of Walter Atkinson, the newly elected secretary, on a colored reflex from the anterior capsule of the lens which occurs in mercurialism. Hunter McGuire of Winchester, Virginia, was elected president, and John Green of Saint Louis, vice-president. The Howe Medal for distinguished service to ophthalmology was presented to E. V. L. Brown of Chicago by the spokesman, Edward Jackson. The pleasant companionship and leisure of this meeting is one of its most attractive features, and the members and guests thoroughly enjoyed the mélange of scientific work and social activities, far removed from thoughts of war for a few days.

On Tuesday, June 9th, the Association for Research in Ophthalmology met at

the Marlborough-Blenheim Hotel in Atlantic City. One hundred and twenty-one members and guests registered. Cecil O'Brien resigned as secretary-treasurer after having played a large part in the success of this organization since its inception 14 years ago. Brittain F. Payne of New York succeeded him. The eight papers on the program covered a variety of subjects. Three of them, ably presented, comprised discussions of the canal of Schlemm and the outflow of aqueous, and opened new investigative vistas. The importance and influence of the Association is necessarily increasing yearly and it fills an important gap in our meetings. Refugee ophthalmologists contributed to half of the program. The mature work of these trained investigators repays the debt owed in being welcomed to our shores and we are the gainers thereby.

That evening the American Board of Ophthalmology gave a military dinner to its members and 60 of the departmental heads throughout the country. The dinner was presided over by William Benedict. Among the speakers were Colonel Leonard G. Rowntree, M.C., U.S.A., Chief, Medical Division, Selective Service System, and Commander Clifford A. Swanson, M.C., U.S.N., Ophthalmologist, United States Naval Medical School, Washington, D.C. They spoke on the need for trained medical officers and how this need is to be met. Those present pledged themselves and their departments to assist in every possible way and to fulfill any demands made upon their facilities and resources in order to provide postgraduate instruction according to military and naval needs.

On Wednesday morning, June 10th, the Section on Ophthalmology held its opening session. Lawrence T. Post presided. Four hundred and sixty-two members and guests registered with the Section. The guest of honor was Moacyr E. Alvaro of São Paulo, Brazil, who spoke on "The effects, other than anti-infectious, of the sulfonamides on the eye." There followed a symposium on geriatrics and its relationship to ophthalmology. The medical, urologic, and neuro-psychiatric aspects were discussed.

That evening a special executive session was held. The relatively poor attendance was disappointing. After much discussion, firmly held in bounds under the able direction of Chairman Post, the Section voted to rescind the resolution of 1941. In other words, the 1935 resolution having to do with optometric relationship stands like an oak.

The scientific session continued until June 12th, when it was adjourned by Chairman Post with the remark "Go ye, and rescind no more." Sixteen papers were read and discussed. The splendid program was somewhat spoiled, indeed nearly ruined, by the devastating physical handicaps of the meeting hall. The loudspeaker system was particularly disappointing, and the competition with the noises on all sides from without the hall was enervating. The climax came at noon each day when the organ located in the main auditorium accompanied the last speaker on the morning's program with semipopular music and arias from operas.

The Section was represented by 11 scientific exhibits, all of high quality and interest. The importance of this feature as a contribution to the success of the meeting cannot be overemphasized. The members sincerely appreciated the efforts of their colleagues in demonstrating and exhibiting the results of their work.

Conrad Berens of New York was elected the new chairman, and Robert Von der Heydt the new vice-chairman. The Section's Research Medal was

awarded to M. Uribe Troncoso for his many notable contributions to ophthalmic research. It is particularly pleasing that the Medal was awarded Dr. Troncoso this year when the American Medical Association had planned a Interamerican convention. The executive committee's choice was exceedingly popular with the members of the Section as attested by the applause. Dr. Troncoso unfortunately was not present on Friday to receive the medal, and the chairman-elect was designated to bestow it in person.

The spring meetings provide enough scientific food to digest for months to come. What the future holds no one can foresee, but at least those of this year were successful and fruitful.

Derrick Vail.

THE PASSING OF A FRIEND

Twenty-four hours before his sixty-second birthday, William P. Wherry died in Omaha. And with his death the sciences of ophthalmology and otolaryngology and those who practice within those realms lost a friend. Organized medicine lost one of its greatest exponents, and the ranks of the progressive leaders of medicine were decreased by one. It was a grievous loss to all.

Previous to 1920, Dr. Wherry had confined his activities to his own community, although he attended the meetings of the national societies assiduously. In that year, he was appointed secretary of the newly formed Section on Instruction of the American Academy of Ophthalmology and Otolaryngology, and his genius for organization and administration was given its first national opportunity. He soon outgrew committee work and only a few years later was elected secretary-treasurer of the Academy, which, under his guidance, forged ahead into the outstanding position that it now occupies. He plunged heart and soul into reorganizing and systemizing the muddled affairs of that organization, in which work he was able to surround himself with a secretarial group. Although this group changed with the years, it has always been dominated by a spirit of harmony and of close coöperation, and by an intense loyalty to the Academy, all due to the guiding influence of Dr. Wherry.

Equal to his love for the Academy was his love for the American Board of Otolaryngology, founded largely through his insistence and organized splendidly by his genius. As secretary of that Board since its inception, Dr. Wherry has been a dominant figure in the specialty boards of this country and as such has had an enormous influence upon the development of the high standards of graduate teaching in the specialties. But despite his national prominence, he never forgot his own community, and the Nebraska State Medical Society honored him by electing him president of that organization for the last year of his life.

Above his love for organization work, for teaching, and for helping the other fellow, Dr. Wherry was first and foremost a physician, with all that that implies. He was never too busy at medical meetings to listen to the discourses and to participate in the discussion, and the scientific exhibits held a fascination for him. He was thoroughly conversant with the modern literature, not only of his specialty, but also of general medicine. That somewhat rare combination of ability, knowledge, and personality attracted a large private practice from the entire Middle West and his passing left a host of loyal grieving patients.

It is unusual in a medical man to find the executive and administrative ability, nay genius, that Wherry possessed. Had he chosen commercial fields instead of the

scientific, he would have ranked among the industrial leaders of America. But he was a physician at heart, and he refused the offer of a great railway system to take a place among its administrative leaders. He preferred to work with and among his fellow medical men and freely gave of his talents that they might be served the better. High in his code was self-effacement and the personal pronoun played but a small role in his vocabulary. All of these traits commanded the real love and unswerving loyalty of those who worked with him and aided him toward achieving his aims of greater opportunity for his fellow practitioners of America, of higher ideals, and consequently of greater service toward the public.

Bill Wherry is going to be missed, sadly missed, not only by those with whom he was intimately associated but equally by those who benefited from his love of mankind. His life has not been lived in vain; he left his mark, written in unfading ink, a mark as a physician, as an educator, and as an idealist to whom future generations of medical men will be beholden.

Harry Gradle.

MUSTARD-GAS INJURIES

Mustard gas, as a weapon of war, was first used by the Germans in 1917. Since that time it has been occasionally responsible for industrial injuries to sight. Eyes so injured have seemed to recover from the injury in a few days, or weeks. The symptoms of irritation and inflammation would subside. The eyes, with relatively slight loss of vision, would be able to perform accustomed tasks. But, as years went by, such eyes, without any active renewal of symptoms, have shown impairment of vision. Such impairment has been found to be caused by progressive clouding of the cornea. Dr. Ida Mann and P. D. Pullinger have presented to the Section on Ophthalmology of the Royal Society of Medicine, in London, the results of a study of mustard-gas lesions in the eyes of rabbits and of men.

Their experiments were made on 101 eyes of rabbits. The chemical was applied to the cornea, in groups of very small dots. Generally the animal had been placed under a general anesthetic; but, without this, they gave very little evidence of pain. Contact of two or three seconds was sufficient to destroy the corneal epithelium, and in six or seven minutes it provoked localized hyperemia of the iris, which generally involved the whole iris. After some hours the eyes looked watery, and there was some hyperemia of the conjunctiva, but no visible change in the corneal tissue on the first day. On the second day the corneal tissue was losing its transparency. After seven days a silky change was noticed in the corneal stroma. Applied to the corneoscleral junction, a corresponding sector of the cornea was affected, with destruction of epithelium, staining and bedewing of an adjoining area, but not of the whole cornea. In 16 days the cornea had apparently recovered; except that there still remained, the "silky" appearance of the corneal stroma.

In severe lesions, there were marked changes in the blood vessels; great irregularities in the caliber of the vessels; some parts greatly narrowed, and others widely dilated. There were some hemorrhages and "blood islands" that remained for months. Necrosis of the cornea and panophthalmitis occasionally occurred in two or three months. The sequels to the experimental injury of rabbits' eyes were followed for two or three years.

From the records of Moorfields Hospital and from other sources the subsequent histories of 39 patients were followed, from the time of injury over periods of up to 15 years. It is concluded

that, allowing for the differences in the length of the life cycle, changes occurring in the rabbit very closely parallel those observed in man. The reaction in man and in the rabbit was dependent on the size of the dose and the anatomic situation. Effects of the vapor and the liquid differ only with the amount of mustard gas that leaks into the tissues. The late keratitis seen in man is a degenerative ulceration, depending on the original damage sustained by the cornea and limbus. "In the present state of our knowledge prophylaxis is likely to be much more important than treatment."

The experiments on rabbits and the lengthened history of the first-war injuries seem to teach that complete and prompt cleansing of the injured cornea is of great importance. Good vision, restored for the time, may be lost by later degenerative changes. These are the two things to be borne in mind with regard to injuries to sight by mustard gas. The late degenerative changes are more apt to appear when the blood vessels of the limbus have been seriously damaged.

The illustrations that accompany this paper include 20 drawings and 6 colored-plate reproductions. They make mustard gas lesions of the eyes a very definite addition to the literature of ophthalmic medicine and surgery.

Edward Jackson.

OUR FRIEND THE QUACK

In many sections of the United States, the prevailing system of elementary and high-school education has not gone very far toward teaching the facts and principles of anatomy and physiology. For the benefit of the rising generation, every schoolteacher ought to be well versed in the essentials of these two related subjects. Every schoolchild needs to be taught enough concerning the human body to prevent his falling an easy prey to the grosser misconceptions so commonly encountered in the physician's contacts with the lay mind.

Such information as the average layman possesses on health and disease depends in part upon the scraps of informal instruction conveyed to him by his physicians, partly upon the often erroneous teachings of periodical literature, partly upon doctrines or traditions handed down from one generation to another.

A few centuries ago the distinction between regular physician and quack was not always very obvious, even to the experienced observer. "In the feudal ages," says Jeaffreson ("A book about doctors"), "medicine and quackery were the same, as far as any principles of science are concerned. The only difference between the physician and the charlatan was, that the former was a fool and the latter a rogue."

About the time of Napoleon, the English clergyman and scholar Samuel Parr offered a definition which may still serve. "The term quack," he said, "is applicable to all who, by pompous pretences, mean insinuations, and indirect promises, endeavor to obtain that confidence to which neither education, merit, nor experience entitles them." The definition is curiously illustrative of the vagueness of many definitions, for each of its clauses might be applied here and there to physicians who are not commonly considered as quacks but who display in some degree similar defects and exaggerations.

Quacks have long been patronized in high classes. "Queen Anne's weak eyes caused her to pass from one empiric to another. . . . She had a passion for quack oculists; and happy was the advertising scoundrel who gained her Majesty's favor with a new collyrium."

Mesmer, who magnetized his patients, took his M.D. degree in Vienna after an

inaugural dissertation on "The influence of planets upon the human body." His English follower Graham (also M.D. but of Edinburgh), was patronized by the Duchess of Devonshire of his time, who is said to have been equally favorable to "every other quack who came her way." Those whose dominant professional qualifications have been learning, modesty, and integrity are sometimes disappointed and incensed at the extent to which a gullible public accepts the ministrations of irregulars, who possess neither education, merit, nor the right sort of experience. We have all been shocked by learning of cases in which the quack was appreciated and we were scorned.

Yet the world does move. Relatively, the enlightenment of the general public today, as compared with two hundred years ago, has probably improved as much as that of the medical profession. Most of the laity do possess some ability to discriminate between true and false practitioners of the healing art.

Particularly are human ignorance and human weakness displayed in matters relating to correction of refractive errors. We are all acquainted with not only women but men who would rather do anything else than wear glasses, and who are willing or anxious to accept the advice of any quack who announces himself a disciple of a school which promises "sight without glasses."

On the one hand we have the mountebank who, ignorant of ocular anatomy and ocular principle, declares in print and in his office that strain, far-sightedness, presbyopia, and cataract are different stages of the same thing, that he has never had a case of cross eyes that has not been improved, and that hyperopia is caused by tenseness of the oblique muscles and myopia by tenseness of the recti.

But on the other hand, the basic support for such a gentleman is to be found in the patient who fears that spectacles will spoil his or her looks, and who will go to any length to escape the wearing of correcting lenses.

After all, are there not a few supposedly orthodox ophthalmologists whose reward of virtue is derived from satisfying their patients, or their patients' parents, that the demands of nature for relief from eyestrain may be ignored and that glasses may be replaced by "exercises"?

Nor may we forget that the activities of the "sight without glasses" specialist are encouraged by poor refraction work, on the part of either physician or optometrist, and that the patient who has never found comfort in glasses is often inclined to discard them forever.

It is noteworthy that the "sight without glasses" specialist does not make his professional fortune by merely telling his clients to throw away their spectacles, or even by prescribing the "basic principles" of "blinking," "shifting," "central fixation," "the long swing," "palming," or sun gazing. He carries up his sleeve some financial trumps of a positive character. Diets are to be recommended, the patient may be persuaded to indulge in the luxury of colonic irrigations, or may be submitted to treatments for anemia or nervousness, or he may be referred to an osteopathic rhinologist or roentgenologist. No one has ever seen a quack who did not mix some common sense with his foolishness. (It is perhaps also true that there are relatively few physicians who do not occasionally mix a little foolishness with their medical wisdom!)

Faddists associate with faddists. The Bates who advised people to throw away their glasses became associated with a notorious "physical-culturist," who cures cancer with milk, and who in turn dubbed Bates "one of the world's leading eye specialists."

The intelligent layman is often capable of appraising such cranks at their proper value. They have their special following —enough occasionally to make them rich in this world's goods. But their obvious inconsistencies, overstatements, and all-inclusiveness betray them, and their position in the general community is always more or less insecure, illicit, and undignified. Their scientific heresy wins them the approval of the ignorant and gullible, but the derision of the enlightened and clear-minded.

In the long view, quackery, for all the mischief it does, detracts in no degree from the esteem in which the general public holds the physician. It has been said that hypocrisy is the homage paid by vice to virtue. In somewhat analogous fashion, the well-trained, intelligent, and honorable physician shines by contrast with the incompetence, cupidity, and chicanery of the quack. Thus, by one of those curious compensations in which life abounds, the quack is in a sense the physician's friend.

W. H. Crisp.

BOOK NOTICES

TRANSACTIONS, SECTION ON OPHTHALMOLOGY, AMERICAN MEDICAL ASSOCIATION, 1941. 382 pages. Illustrated. Chicago. Printed by the American Medical Association.

The need for recognized standards of medical ethics led to the organization of the American Medical Association 100 years ago. The chairman's address at the 1941 meeting of the Section on Ophthalmology, by Dr. Albert C. Snell, is entitled, "Some principles of medical ethics applied to the practice of ophthalmology." It is a real contribution to practical ophthalmology.

The paper of Dr. A. J. Bedell, of Albany, New York, deals with "Traumatic changes in the retina, choroid, nerve head, and vitreous," as these have been studied by photography of the ocular fundus. Dr. Bedell has worked up his subject so well that even without his pictures, his clear thinking makes a valuable addition to any ophthalmic library.

Dr. Albert A. Brown, of Cincinnati, makes a contribution to practical ophthalmic surgery, in his paper on "Lime burns of the eye. Use of rabbit peritoneum to prevent severe delayed effects." Lime burns were encountered long before mustard gas was used in warfare. But the management of such injuries, to prevent the later defects of the cornea, now recognized, is a timely addition to our therapeutic resources, and demonstrates that rabbit peritoneum answers the needs for a protective membrane. The paper is well illustrated.

Drs. G. G. Gibson and L. W. Smith, of Philadelphia, have made a study of the "Pathology of retinal phlebosclerosis." It illuminates a relatively obscure subject in ophthalmic practice. "The retina in systemic vascular hypertension" is the subject of a paper by Dr. Ferdinand L. P. Koch, of New York, that will be found suggestive and valuable. "The causes of blindness in Pennsylvania" is an analysis of the blindness in over 30,000 eyes, made by Dr. Alfred Cowan and Bernice C. English. Senility is given as the cause in 9,238; syphilis in 1,843, followed by cardiovascular disease in 1,795. "The etiology of uveitis. A clinical study of 562 cases" by Drs. Jack S. Guyton and Alan C. Woods, of Baltimore, would interest any gathering of ophthalmologists.

Other papers published in this volume have positive value for the studious practitioner of ophthalmology. Burian has worked out some new methods of observing ocular movements in permanent strabismus. Berens and Nilson report on ocular conditions associated with coliform

bacterial infections. Harrington and Parsons discuss the standardization of tonometric observations. Thygeson and Stone have a paper on epidemiology of inclusion conjunctivitis, and W. T. Davis one on superficial punctate keratitis. T. D. Allen and W. G. Ackerman report on hereditary glaucoma, in three generations; and J. W. White on paralysis of the superior rectus and inferior oblique muscles of the same eye; S. G. Higgins on a corneal-scleral suture, he has used for eight years.

For practical value, it is doubtful if any annual volume on ophthalmology records more of active scientific progress. The list of Fellows of the Association, registered in the Section on Ophthalmology, gives a very complete list of American ophthalmologists. The record of their attendance can be used as an indication of the interest of each in his professional work. Last year there were 242 members registered as present; but the list shows that 255 subscribe for its Transactions. The broader circulation of the Transactions fully justifies the publication policy of the American Medical Association. Interest awakened by hearing a paper read is most profitable when it leads to the careful study of it.

Edward Jackson.

MERRILL'S EYE NOTES, The Mid-Winter Clinical Course given by the Research Study Club of Los Angeles. By H. G. Merrill, M.D. Illustrated (sketches). 393 pages. Printed by the Webbcraft Printers, Los Angeles, 1941. Price $27.00.

In his foreword the author states that the notes were made mostly by himself during the Mid-Winter Clinical Course in Ophthalmology at Los Angeles from 1932 through 1937. They were then printed and illustrated with black-and-white sketches on good quality paper, and bound in a leatheroid loose-leaf binder. As an informal record of the remarks of distinguished guests of the course throughout these years, the notes contain many items of interest and value. The author mentions that there was no editing of the notes, and this is obvious on reading them. Much could have been eliminated, without loss; other material could have been elaborated upon. Each lecturer should have had the opportunity of looking over his remarks, not with the idea of changing them, but rather with the purpose of ironing out the rough places, correcting obvious mistakes and wrong impressions without sacrifice of informality. The lectures are not dated, although it would be of interest to know when Drs. Elschnig, Bielschowsky, and Robert E. Wright, for example, lectured. The lectures by Meyer Wiener were edited by himself and glow with the speaker's personality and teachings. They form one of the most important and attractive sections of the work.

There is undoubtedly a place for this type of ophthalmic literature, as Edward Jackson pointed out in an editorial in these pages (issue of November, 1941). It requires, however, careful treatment to be of value to any but the author himself, whose memory can be jogged by a few remarks, whereas the outsider reading them "cold" needs more elaboration.

The eye notes as presented are much too expensive, and needlessly so. Mimeographic or lithographic copies would have delivered the same message more cheaply. As it is they constitute a "collector's item" and are not for the purse of the average student of ophthalmology.

Derrick Vail.

THE EXTRA-OCULAR MUSCLES, AN OUTLINE. By Wendell L. Hughes, M.D. Second edition. Paper

covers, 32 pages, 9 illustrations. Copyrighted 1939. Price $2.00 plus postage.

Bound in loose-leaf style, the pages of which are blank on one side, the publication performs a useful function in furnishing a quick outline review of the essential data of the subject. The blank pages afford space for the student's own notes, and remarks of his collateral reading. No new facts are presented, but the presentation is obviously designed for the running, reading individual and for the student who encounters the extraocular muscles for the first time and wishes review notes. Its value will be enhanced by the number of notes and remarks that the student himself inserts from his outside reading. The teacher will find the outline useful in his class work.

Derrick Vail.

OBITUARY

EMILE DE GROSZ
1865-1941

Emile de Grosz was born in Nagyvarad, Hungary, on September 30, 1865. His grandfather, Frederick (1798-1858), had founded the first charity hospital for eye disease in Europe in 1830, and his father, Albert, was also an eye doctor. After careful early schooling he entered the University of Budapest, then a 300-year-old institution with a five-year medical course, where he became an assistant in anatomy and did special work in embryology. After graduation in 1887, he took a course in public health. The next year was spent in the eye clinics of Vienna, Prague, Berlin, Heidelberg, Paris, and Brussels, and at the end of the year he joined the department of ophthalmology at the University of Budapest as assistant.

At this time the clinic had 53 beds, a small library, a very primitive laboratory, but a huge out-patient department and a fair operating room. There was, however, no provision for private patients, who were operated on at home or in hotel rooms. The chief of the clinic selected Dr. de Grosz for his private assistant; they carried the instruments in a small velvet-lined case and cleaned them in alcohol. For sponges they used small pieces of linen which were boiled and ironed. Professor Schulek, Dr. de Grosz's chief, had one postoperative infection in 30 years of private practice.

Clinical assistants were supposed to live in the hospital and it was with great difficulty that he obtained permission to get married, which he did in 1892, marrying Margaret de Takats. The permission came at the last minute, for he was ready to resign and obtain a position in his home town had his request been refused. This would have meant the end of his connection with the University and would have undoubtedly greatly modified his career. After a short honeymoon he entered the clinical service, starting with a course on operative technique for students at 7:00 to 7:30 P.M. There was no time for private practice, although it was not prohibited.

In 1892, after four years of service at the clinic, Dr. de Grosz found himself in temporary charge. His chief had become ill and two of the senior assistants left for other positions. Thus, at the age of 28 years, he was charged with the systematic lectures and the operative work, although the examinations were still given by one of the older extramural professors. He nevertheless found time for some publications relating to neuroparalytic keratitis and tabetic atrophy of the optic nerve. He also started to frequent the meetings of the international Ophthalmologic Congress, and gave two papers in Utrecht in 1899. His friendship with Argyll Robertson, George Berry,

Pristley Smith, Treacher Collins, Haab, Panas, Leber, Sattler, and many others dated from these early, truly international gatherings.

In 1894 his chief returned, and de Grosz was put in charge of the laboratory. Because of its vast material this was a veritable treasure for men interested in the pathology of the eye. Young eye surgeons from all over Europe came to work here and later this laboratory became well known to American investigators as well.

During his assistantship he developed the form and color tests for railway engineers subsequently widely adopted. An associate professorship and charge of 53 beds in a municipal hospital came in 1900, and headship in the University eye department in 1904. At that time trachoma was a major cause of blindness in Hungary and as State Commissioner he established hospitals that decentralized care and secured a type of legislative control that became a model for many other countries. Under his leadership (1904-1936) the department grew and expanded greatly. Beds increased from 53 to 134, and a second chair of ophthalmology was created with a new building housing 120 beds (1907). In 1914 he became Dean of the Medical School.

First charity eye hospital in Europe founded in 1830 by Frederick de Grosz at Nagyvarad, Hungary.

During World War I he formed trachoma battalions in which 75 percent of the recruits were successfully treated. It could then be said that while the army had spread trachoma 100 years before, it helped reduce it greatly in 1914-1918. Later as chairman of the executive committee of the faculty he enlarged the University clinics so that, by 1925, 1,800 teaching beds were available. Among his many papers before European eye societies perhaps the more important are the ones on optic atrophy due to methyl alcohol (1910), the Hunterian Lecture on glaucoma (London, 1939) and his dis-

cussion of sympathetic ophthalmia before the French Society. He was the guest of the American Academy of Ophthalmology and Otolaryngology in 1930.

His activity in medical education extended through 50 years, and culminated in a complete revision of the curriculum in 1922. A comprehensive examination in physics, chemistry, anatomy, and physiology was held at the end of the second year. Examinations in the clinical subjects were held during the sixth year of interneship, each examination following the interneship in a particular field. Such a truly remarkable system could be carried out only in a medical school which can accommodate and supervise its own students and need not farm them out to hospitals that sometimes are not well equipped and maintained.

Postgraduate instruction in Hungary started in the year 1883. Professor de Grosz developed it into a smoothly functioning system and obtained potent financial support from the State. He started and edited a journal, which contained most of the lectures given, and headed the Committee on Postgraduate Education for 28 years. Courses lasting 2 weeks, 4 weeks, or 6 weeks were given. Over 11,000 physicians took part during these 28 years. To facilitate their attendance, a central building, containing 40 rooms, was erected. Board and rooms were provided at cost for the attending groups. If necessary, the committee had funds to reimburse the doctor for traveling and living expenses. This system is still functioning and has no doubt elevated the standards of medical education in Hungary. Later, different groups throughout Europe established an International Committee for Post-graduate Education in the founding and development of which Professor de Grosz took an active part.

Much of his energy was devoted to the organization of international medical congresses. He was secretary of the international medical congress held in London in 1909 and invited the next meeting to convene in Budapest. This occurred in 1913 with 5,000 members participating.

He also took active part in the international congress for public hygiene held in Dresden in 1911, and organized the Hungarian building, which won a great deal of recognition. The early legislative actions of the Hungarian government on child welfare were demonstrated here for the first time.

He was equally active in the Medico-legal Council, an organization that rendered the court's independent medical opinion, the members being appointed by the State. These were nonsalaried positions and were regarded as a performance of public duty. They ruled on the often diametrically opposed medical testimonies.

His life embraced the period of great national development, extended international coöperation, wide-going liberalization and socialization of the early part of the twentieth century, and it is doubtful if any other eye doctor in Europe was more active or important in all this than was Emile de Grosz.

He died on December 8, 1941, in his 77th year. Surviving him are an ophthalmologist son, Stephen, of Budapest, a daughter living in Budapest, and a son living in the United States, Dr. Geza de Takats.

E. V. L. Brown.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

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EYEBALL AND ORBIT

Cotlier, I., and Sanz, J. **Traumatic enophthalmos.** Anales Argentinos de Oft., 1941, v. 2, June, pp. 75-84.

After reviewing Birch-Hirschfeld's series of cases, the author reports two of his own. The first was in a man of 56 years, who was struck on the head with a stick. He developed a severe orbital hemorrhage, followed later by resorption of the retro-ocular fat and fixation of the eyeball by scar tissue with diminished ocular motility. The second case was in a workman aged 29 years, who in starting a motor sustained a fracture of the superior maxillary bone and a hemorrhage within the maxillary sinus. (6 illustrations, references.) W. H. Crisp.

Dixon, G. J. **Unilateral exophthalmos.** Brain, 1941, v. 64, March, pp. 73-89.

Proptosis with lateral or vertical displacement of the globe usually indicates the presence of some abnormal structure in the orbit outside the cone of ocular muscles. Symmetrical proptosis may be classified for diagnostic purposes into that showing papilledema of the proptosed eye, that with abnormality of internal musculature or gross abnormality of external movements, and that with weakness of the external ocular muscles but no involvement of the ocular reflexes or vision. The symmetrical cases may be due to tumors of the optic nerve, lymphatic and venous engorgement of the orbit, or paresis of the ocular muscles. Seventeen cases illustrating points in diagnosis are presented. John C. Long.

McGregor, I. S. **Orbital cellulitis from gas-producing organisms.** Brit. Med. Jour., 1942, Feb. 28, p. 292.

A case is reported of a 67-year-old maltster who for five days had had very severe pain in the right eye, while the lids became greatly swollen. Brown pus and bubbles of gas escaped from incisions in the lids. The bacteriologist reported the presence of various cocci and a few Gram-positive bacilli from

a smear, and, on culture, staphylococcus aureus. A later stab culture showed a Gram-positive bacillus with filamentous forms, some of which presented swellings resembling spores. The case was treated by anti-gas-gangrene serum and sulfathiazol. The orbit cleared entirely, but the eye was rendered blind by optic atrophy.

Ralph W. Danielson.

Seitchik, J. N. **Mechanism of the production of exophthalmos in exophthalmic goiter.** Arch. of Ophth., 1942, v. 27, April, pp. 762-775.

A review of the literature shows that it is impossible to select a single local or general cause for exophthalmos at the present time. The author discusses the role of orbital disease, of the sympathetic nervous system, of the thyroid gland, of the anterior lobe of the pituitary, and of the gonads in the production of exophthalmos. He concludes that malignant exophthalmos results from mechanical pressure due to the increased volume of the ocular fatty connective tissue and of the extra-ocular muscles. This results from hypertrophy of the fat and parenchymatous degeneration and fibrosis of the muscles, as well as edema and round-cell infiltration of both structures. It is suggested that post-thyroidectomy progressive exophthalmos is caused by an increased secretion of thyrotropic hormone in patients in the hypothyroid state. The cause of reversible, non-progressive exophthalmos as seen in patients with exophthalmic goiter is not known. J. Hewitt Judd.

14

EYELIDS AND LACRIMAL APPARATUS

Malbran, Jorge. **Operative correction of palpebral ptosis.** La Semana Med., 1941, v. 48, Dec. 18, pp. 1456-1462.

The author discusses the value of various classical operations, particularly the Blaskovics and the Motais. He finds the Blaskovics the operation of choice in the majority of cases, especially those of moderate severity. In complete ptosis, if the superior rectus has full functional power, he advises Wheeler's operation, although here also the Blaskovics is effective, supplemented where necessary by de Lapersonne's procedure. (5 illustrations.) W. H. Crisp.

Moret, R. L., Damianovich, J., and Vidal Freyre, A. **Conjunctivitis in the nursing child from lacrimal obstruction.** La Semana Med., 1942, v. 49, Feb. 26, pp. 419-421.

The authors report 14 cases. They remark that the obstruction may consist of simple lack of perforation of the lacrimal punctum or the lacrimal canal in the inferior meatus of the nasal fossa, or that it may depend upon the presence of a small plug of epithelial desquamation. In the latter event, it is sometimes sufficient to irrigate the lacrimal passages once or twice with physiologic-salt solution under pressure. In other cases the use of probes is necessary. Occasionally it is sufficient to dilate the lacrimal punctum.

W. H. Crisp.

Morgenstern, D. J. **Intranasal drainage for cure of chronic tear-sac infection.** Arch. of Ophth., 1942, v. 27, April, pp. 733-745.

This report is based on three cases, in each of which a permanent cure was obtained by making an opening through the bone into the tear sac by means of electrocoagulation. A dental root-canal probe is passed through the punctum and canaliculus until the tip reaches the medial wall of the sac.

Tipping it downward until it reaches the bottom of the sac it is then forced through the lacrimal bone into the nasal cavity at an angle of 45 degrees. Specially prepared hooked instruments are used to enlarge the opening to a vertical oval 4 by 12 mm. The tip of the electrode is then rotated two or three times around the opening to destroy fragments of the torn medial wall, and the sac is irrigated with warm physiologic-salt solution each week. Electrocoagulation is repeated after two weeks to insure an adequate opening. This procedure gives a permanent small fistula, oval in shape, about 2.5 by 3.5 mm. When the sac is filled with granulations or scar tissue or is badly diseased, electrocoagulation destroys the contents and the mucous-membrane lining of the sac wall and to a lesser extent the sac itself. This leads to regeneration of the mucous membrane with the least possible tendency to granulation. When indicated, complete removal of infection is accomplished by retrograde electrocoagulation of the nasolacrimal duct. Because of the slightness of the surgical intervention and the subsequent coagulating action of the electric current, bleeding and possible infection both operatively and postoperatively are reduced to the lowest possible degree.

J. Hewitt Judd.

Pereira, R. F. **Hess operation.** Arch. de Oft. de Buenos Aires, 1941, v. 16, May, p. 241.

This paper deals with the types of dressing applied immediately after the operation of Hess. The author strongly favors Meller's bandage, using a piece of transparent celluloid, fastened with adhesive, to cover the eye. This dressing, which allows daily observation of the eye, is changed when conjunctival secretion is present.

Plinio Montalván.

Rosenvold, L. K. **Dacryocystitis and blepharitis due to infection by aspergillus niger.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 588-589.

15

TUMORS

Basora Defillo, Juan, and Laugier, A. R. **Retinoblastoma.** Bol. de la Asoc. Med. de Puerto Rico, 1942, v. 34, Feb., pp. 55-58.

Three cases are reported, all with fatal outcome. Two cases showed extension to the brain, and one had metastases in both femurs, the superior maxillary, and the ribs, liver, and lungs.

W. H. Crisp.

Bellecci, Paolo. **Contribution to the clinical and anatomopathologic study of basal-cell tumors of the lids.** Ann. di Ottal., 1940, v. 68, Nov., pp. 863-872.

A basal-cell epithelioma developed from the lid margin of a sixty-year-old woman, who had noted the first sign of the tumor about two years previously. Two small portions were removed for biopsy. The histologic structure is described in detail. Treatment with X rays resulted in a clinical cure: six months later there had been no recurrence. The literature is reviewed at length.

Harry K. Messenger.

Bothman, L., and Blankstein, S. S. **The eye in adrenal sympathicoblastoma (neuroblastoma).** Arch. of Ophth., 1942, v. 27, April, pp. 746-761.

Four cases of adrenal sympathicoblastoma with ocular manifestations are reported in children (three male and one female) aged 2, 2½, 3½, and 5 years. Typical findings are illustrated

by photographs and photomicrographs. Ecchymosis of the eyelids was the most constant finding, occurring in all four cases. Papilledema occurred in two cases, exophthalmos in one, a temporal tumor in one, and paralysis of the external rectus muscles in one. The importance of ecchymosis and exophthalmos as distinct aids to diagnosis is stressed, since biopsy often fails to provide the correct diagnosis. The overlapping of the Pepper and Hutchison types, which are differentiated solely on the site of major metastases, is well illustrated by the cases reported, all of which were of the Hutchison type. The fact that this tumor metastasizes to the globe by way of the blood stream was demonstrated by the presence of typical tumor cells in the medium-sized blood vessels of the choroid and the choriocapillaris. A review of the literature reveals 78 cases having one or more ocular signs. These clearly show that the occurrence of ecchymosis alone or with exophthalmos may be diagnostic of adrenal sympathicoblastoma, because other causes of ecchymosis of the lids, such as chloremia, leukemia, purpura, scurvy, and trauma, may be excluded by the history or by laboratory examinations. (Bibliography.)

J. Hewitt Judd.

Calhoun, F. P., Jr., and Reese, A. B. **Rhabdomyosarcoma of the orbit.** Arch. of Ophth., 1942, v. 27, March, pp. 558-578; also Trans. Amer. Ophth. Soc., 1941, v. 39, p. 139.

The authors review 14 cases found in the literature and report five additional cases. In the series from the literature, in which all but one of the patients were young children, the course was characterized clinically by rapid growth of the tumor, and in five cases followed, death occurred within 1½ years after appearance of the tumor. In only one case was the growth said to have been arrested for any length of time, the period in this case being 2½ years after exenteration. All five of the authors' patients received preliminary irradiation, which was without effect on the growth except in one patient in whom there was temporary clinical regression for 15 months. Of the five patients, two were without evidence of recurrence two years after exenteration of the orbit and more than three years after the first appearance of the tumor. One patient was without evidence of recurrence one month after exenteration of the orbit. One patient died five months after exenteration of the orbit. In one patient the tumor developed behind the remaining eye after enucleation of one eye and intensive irradiation to the other for bilateral retinoblastoma seven years previously, suggesting that it may have been induced by irradiation. The apparent arrest of the growth in two cases suggests that this type of orbital sarcoma has a more favorable prognosis than that generally assumed for sarcoma of the orbit in general. (Photographs, photomicrographs, bibliography.)

J. Hewitt Judd.

Ch'in, K. Y. **The histogenesis of glioma retinae.** Amer. Jour. Path., 1941, v. 17, Nov., p. 813.

A case report of retinoblastoma originating from the inner nuclear layer and still confined within the limiting membranes of the retina.

Theodore M. Shapira.

Foroni, Gian Maria. **Clinical and histopathologic contribution to the study of the behavior and evolution of sarcomas of the papillary region, with**

particular regard to the ways of extrabulbar propagation. Ann. di Ottal., 1940, v. 68, Oct., pp. 771-791.

A case of melanosarcoma of the papillary region is reported. The histologic structure is discussed in detail. Although gross inspection of the enucleated eye disclosed no evidence of extraocular extension, serial sections showed that the tumor had extended outside the eye. Melanosarcomas at the posterior pole are particularly dangerous because they have a ready means of extraocular extension in the numerous passageways of the posterior ciliary arteries and the ciliary nerves. Hence in such cases it is important to study the serial sections.

Harry K. Messenger.

Gözberk, Rifat. **Epibulbar dermolipoma.** Türk Oft. Gazetesi, 1941, v. 3, pt. 9, p. 425. (French abstract, p. 443.)

A case report.

Gözcü, Niyaze. **Subconjunctival dermolipoma.** Türk Oft. Gazetesi, 1940, v. 3, pts. 7-8, p. 345. (French abstract, p. 398.)

Case report of a 13-year-old girl who had had a small epibulbar tumor since birth. This was removed and examined microscopically. George A. Filmer.

Guibor, G. P. **Adrenal neuroblastoma with orbital metastases.** Amer. Jour. Ophth., 1942, v. 25, April, pp. 387-397. (5 figures, references.)

Leite, Olive. **Tumors of the eyeball.** Ophtalmos, 1941, v. 2, no. 2, pp. 242-255.

A girl of 13 years had a melanosarcoma between the cornea and the caruncle of the right eye. It was excised, and the base cauterized with the galvanocautery. A woman of 48 years had an angiosarcoma of the conjunctiva and of the upper culdesac, which had existed since infancy but had developed rapidly in the preceding eight months. A lipoma was removed from the right eye of a boy aged 17 years. It had existed since early childhood and had grown to the point of protruding between the eyelids. (9 illustrations, bibliographies.) W. H. Crisp.

Malbran, J., and Tosi, B. **Papilloma of the conjunctiva.** Arch. de Oft. de Buenos Aires, 1941, v. 16, May, p. 243.

The authors report two cases of papilloma of the conjunctiva and present a thorough review of the literature concerning this condition. They conclude that this type of ocular tumor is not so benign as is generally believed, and they advocate complete surgical excision, followed by radiotherapy if the biopsy is inconclusive regarding the histopathologic nature of the growth. Plinio Montalván.

Marshall, Don. **Malignant melanomas of the eye and adnexae.** Trans. Amer. Acad. Ophth. and Otolaryng., 1941, 45th mtg., March-April, p. 98.

This is a study of an unusual series of cases of conjunctival nevi from which the author concludes that such lesions are rather common and sometimes overlooked. They may be or may become highly malignant, but are ordinarily benign. (9 figures, discussion.)

George H. Stine.

Nordmann, Jean. **Tumors of the ciliary body.** Ophthalmologica, 1941, v. 102, Nov., p. 257.

After a brief review of the normal structure of the ciliary body, the author gives a systematic account of the tumors of this organ. All such tumors consist of rows or tubes of cells. The

benign tumors are either of the inflammatory-pseudotumor type or the Fuchs type of epithelioma. The latter develop in cysts of the senile ciliary body and the cells resemble those seen in certain hyperplasias. The malignant tumors may be placed in one of two groups, depending on whether the cells still retain their maximum potential of development. In those of the adult type, the malignant character is manifested by invasion of the ciliary body. The tumors of the embryonal type are neuroepitheliomas or medullo-epitheliomas like those often found in the central nervous system.

F. Herbert Haessler.

Rocha, H., and Elejalde, P. **Regarding the classification of retinal gliomas.** Ophtalmos, 1941, v. 2, no. 2, pp. 294-307.

The subject is discussed briefly, with 15 illustrations and specific reference to two cases studied clinically and microscopically. W. H. Crisp.

Terrien, Félix. **Flat choroidal sarcoma.** Arch. d'Opht. etc., 1939-1940, v. 3, no. 11, p. 967.

A 56-year-old man became alarmed by a slight haziness in his right upper visual field. An immobile flat retinal detachment was found. No tear was present, and scleral transillumination failed to produce a bright pupil when applied to the lower half of the sclera. Enucleation was performed and histology confirmed the clinical diagnosis. The author states that his case resembled that of Ciotola (Amer. Jour. Ophth., 1939, v. 22, p. 1310). (Review of the literature, photomicrographs.)

K. W. Ascher.

Tiscornia, B. J., and Lami, I. **Epithelioma of the conjunctiva, radium therapy.** Arch. de Oft. de Buenos Aires, 1941, v. 16, Feb., p. 55. (See Amer. Jour. Ophth., 1941, v. 24, Nov., p. 1337.)

Villegas, R. R., Damel, C. S., and Molinari, P. C. **Glioma of the orbit.** La Semana Med., 1942, v. 49, Feb. 19, pp. 354-359.

A woman of 24 years had had two previous operations, the second using the Krönlein technique. Applications of deep radiotherapy were also reported. The eye, which was blind, protruded to such an extent that suture of the eyelids was necessary to prevent a corneal lesion. The orbit was exenterated. Microscopic study of the tumor, which with the eyeball measured 6 by 5 cm., showed a mixture of a glial type of cell with small cells. (8 illustrations, references.)

W. H. Crisp.

16

INJURIES

Bendel, A. J. **Traumatic changes in the retina, choroid, nerve head, and vitreous.** Jour. Amer. Med. Assoc., 1942, v. 117, Nov. 22, p. 1774.

In view of the increasing number of ocular tragedies in industry, in the home, in the school, and in travel, the author describes the many ophthalmic lesions in detail. He concludes that an ophthalmoscopic examination is imperative in every case of injury to the head or the eye. (Discussion.)

George H. Stine.

Biederman, J. B. **A preliminary report on a new eye-particle remover.** Jour. Amer. Med. Assoc., 1942, v. 118, March 7, p. 802.

The appliance consists of a plastic magnifier and a magnetized wire loop.

The particle is located by the magnifier and then removed by the loop.

George H. Stine.

Correa, C. **Sensorio-sensitive-motor traumatic ophthalmoplegia.** Ophtalmos, 1941, v. 2, no. 2, pp. 428-442.

A man of 27 years, riding in an omnibus, was injured by having the end of a bamboo driven into his left orbit. He showed complete loss of vision of the left eye, paralytic mydriasis, ocular immobility, corneal anesthesia, painful anesthesia of the area supplied by the region of the face innervated by the three branches of the trigeminus, and loss of taste on the left half of the tongue. The first supposition of penetration through the ring of Zinn was abandoned in favor of a fracture of the orbit radiating to the orbital apex and floor and involving the round and oval foramina. After four months, functional recovery was limited to the area supplied by the oculomotor. (8 illustrations, references.) W. H. Crisp.

Fisher, H. E. **Electrical accidents: shock, burns, and glare injuries to the eyes.** Arch. Phys. Therapy, 1941, v. 22, Oct., p. 611.

A discussion of accidents by the chief surgeon of the Chicago Rapid Transit Company, with an earnest plea for preventative measures by education. In 29 years of observing glare injuries Hart says he has not seen one patient with permanent visual damage.

Theodore M. Shapira.

Gallo, A., and Toledo, C. de. **Ophthalmia from frog poison.** Ophtalmos, 1941, v. 2, no. 2, pp. 393-401.

A boy of six years, with other children, had been throwing stones at a frog. The mother stated that the frog had "urinated" in the child's eyes! When brought to the ophthalmologist three days later, the child showed severe hyperemia of the palpebral and bulbar conjunctiva of the right eye, the cornea was moderately inflamed, and the pupil was dilated and the light reflex diminished. Recovery occurred under a simple collyrium. The toxic action, in certain poisonous species, appears to arise from special glands which are distributed over the back and in other parts of the animal. (10 illustrations, references.) W. H. Crisp.

Gördüren, Süreyya. **A case of ocular contusion with various tears of the iris.** Türk Oft. Gazetesi, 1941, v. 3, pt. 9, p. 411. (French abstract, p. 442.)

Following a blow on the closed eye there developed ciliary injection, irregular mydriasis, and hyphema. Numerous dialyses, tears, and fissures in the iris were also noted.

George A. Filmer.

Gözberk, Rifat. **Foreign body in the orbit causing meningoencephalitis.** Türk Oft. Gazetesi, 1941, v. 3, pt. 9, p. 423. (French abstract, p. 443).

A small boy suffered a fall, and a stick he was carrying penetrated the orbit. A violent streptococcic and pneumococcic meningitis developed, causing his death. George A. Filmer.

Hughes, W. F., Jr., **Mustard-gas injuries to the eyes.** Arch. of Ophth., 1942, v. 27, March, pp. 582-601.

This review includes a discussion of the incidence and dose necessary to produce a lesion, the course of severe corneal burns, the local and systemic actions of the gas, prophylaxis, and the treatment of the patients without corneal involvement and with corneal involvement. The treatment, which is

largely symptomatic, consists of reassurance of the patient that he is not blind by opening the swollen lids, immediate irrigation of each eye for two minutes with 1.5-percent sodium-bicarbonate solution, physiologic-salt solution, 2-percent boric-acid solution, or 0.5-percent solution of dichloramine-T in chlorinated paraffin, and in cases with corneal involvement or marked photophobia, instillation of 1-percent atropine solution or ointment every day until the symptoms or the iritis subsides. In cases of severe involvement the author advises the instillation of liquid paraffin, cod-liver oil with added sodium bicarbonate and dextrose, or acriflavin in castor oil (1 to 15,000) three times a day, but not sooner than 24 hours after the exposure to the gas. Dark glasses or a brown eye-shade are advisable but should be worn for the shortest possible time in order that the development of neurasthenic symptoms may be avoided. Bandages or eye pads should not be used. Instillation of mild silver-protein solution (10 percent) followed by irrigation with boric-acid solution may be used three times a day to prevent secondary infection. After the initial chemotic and exudative reaction has subsided, the use of zinc-sulphate solution (0.25 percent) with epinephrine is recommended during convalescence. Psychiatric care is often advisable in serious cases to prevent chronic neurasthenia and hysterical reactions.

J. Hewitt Judd.

Kösebay, Baha. **Subconjunctival luxation of the lens.** Türk Oft. Gazetesi, 1940, v. 3, pts. 7-8, p. 393. (French abstract, p. 403.)

The condition resulted from injury by a cow's horn.

George A. Filmer.

Pelner, Louis. **Emergency spud.** Amer. Jour. Ophth., 1942, v. 25, April, p. 456. (One illustration.)

Queiroga, Geraldo. **Considerations on the blue appearance of the eyeground following prolonged use of methylene blue.** Ophtalmos, 1941, v. 2, no. 2, pp. 286-290.

The author had published a case of this kind in Brazil Medico in February, 1939. He refers to previous records by Lambert-Gerber. After reviewing his own case and his previous conclusions, Queiroga expresses the opinion that blue discoloration of the ocular fundus after prolonged use of methylene blue indicates a lowered general vitality, with lack of power to reduce the dye (possibly arising from diminution or absence of glutathione in the vitreous). Cyanochromia of the vitreous is particularly apt to be seen in the antemortem stage in a patient who has been treated with methylene blue.

The author mentions a patient with basal metabolism plus 2, and chronic malaria, who after taking methylene blue for two years did not show the slightest amount of blue staining of the vitreous.

W. H. Crisp.

Smith, H. J., and Wadsworth, H. V. **Nonindustrial eye injuries.** Illinois Med. Jour., 1941, v. 80, Nov., p. 383.

The authors report seven unusual cases of nonindustrial eye injury. They stress the following: careful history, X ray of the eye, local treatment, and tetanus antitoxin in all perforating injuries.

Theodore M. Shapira.

Summers, T. C., and Hobbs, H. **Glass in the anterior chamber.** Brit. Jour. Ophth., 1942, v. 26, Feb., p. 54.

A case is reported in which a spicule of glass was retained for five months

in the anterior chamber. At the end of this time, severe irritation of the eye suddenly developed, with greatly reduced vision. After six weeks of treatment, the cornea was clear enough to permit removal of the spicule of glass. The wound healed rapidly and the vision improved from perception of shadows before operation to 6/5 after operation. Edna M. Reynolds.

Vila Ortiz. **Considerations concerning injuries of the cornea due to industrial accidents.** Arch. de Oft. de Buenos Aires, 1941, v. 16, Jan., p. 19; Feb., p. 70; March, p. 130; and April, p. 180.

The different types of ocular injuries of the cornea due to industrial accidents and their mechanism of production are reviewed. A statistical study and the medicolegal aspects of the problem are presented.

Plinio Montalván.

17

SYSTEMIC DISEASES AND PARASITES

Cordes, F. C., and Hogan, M. J. **Dysostosis multiplex (Hurler's disease, lipochondrodysplasia, gargoylism).** Arch. of Ophth., 1942, v. 27, April, pp. 637-664; also Trans. Pacific Coast Oto-Ophth. Soc., 1941, 29th mtg.

The authors review fifty cases reported in the literature as to etiology, symptomatology, ocular findings, ocular pathologic changes and roentgenologic findings, and report five cases of this syndrome occurring in three families. Two of the patients from different families were twins, and in each case the other twin was unaffected. In all cases both corneas were involved and slitlamp examination, used in four cases, revealed a diffuse infiltration of small, yellowish-gray dots, varying in size, which were scattered throughout the corneal stroma. These corneal infiltrates have been identified as lipid deposits. This syndrome belongs to a group of diseases characterized by disturbances in the storage of lipid. Other members of this group are Nieman-Pick's disease, Hand-Schüller-Christian's disease, Gaucher's disease and Tay-Sachs's disease. The syndrome usually appears at the age of one year and is characterized by bony defects. There is also a wide separation of the orbits and prominence of the globes. (Photographs.) J. Hewitt Judd.

Igersheimer, Joseph. **Ocular disturbances in a case of acromegaly complicated by diabetes.** Arch. of Ophth., 1942, v. 27, Feb., pp. 330-341.

The case reported is that of a man aged 35 years, who presented acromegaly, complicated by diabetes, with retinal lipemia and peculiar visual disturbances, Analysis of the ocular changes indicates that two processes were going on in the pituitary-diencephalic region. One was characterized by bitemporal hemianopsia and was caused by the pressure of a pituitary tumor. The other, causing involvement of the central vision and the visual field as a whole, was located probably in the diencephalic region. These disturbances were favorably influenced by insulin treatment as well as by roentgen-ray treatment. This appears to be the first published case. J. Hewitt Judd.

Malbran, J., and Lima, E. J. **Ocular manifestations of poradenitis.** Arch. de Oft. de Buenos Aires, 1940, v. 15, Oct., p. 524.

In a series of 24 patients affected with lymphogranuloma inguinalis (Nicolas-Favre disease) ocular involvement was found in 82.6 percent of the cases. Frei

tests were positive in all but one case. The most frequent ocular manifestations of the disease are papilledema, peripapillary swelling, and tortuosity with engorgement of the retinal vascular tree, particularly the veins. Visual acuity was consistently normal, and no field changes were observed except a slight enlargement of the blind spot when papilledema was present. No disturbances of the intraocular tension or lesions of the corneal nerves were found in any of the cases. (Bibliography.) Plinio Montalván.

Mendonça de Barros, José. **Biomicroscopic aspects of the ocular complications of leprosy.** Ophtalmos, 1941, v. 2, no. 2, pp. 256-275.

With 24 illustrations, and upon the basis of 1,200 patients seen in the course of seven years, the author describes a group of corneal and iris changes as studied with the slitlamp.

W. H. Crisp.

Oliphant, J. W., Powell, W. F., and Perrin, T. L. **Sulfadiazine in lymphogranuloma venereum ophthalmitis.** Jour. Amer. Med. Assoc., 1942, v. 118, March 21, p. 973.

A patient with probable accidental infection of the eye with the virus of lymphogranuloma venereum was successfully treated by the administration of sulfadiazine, after having resisted all local treatment and foreign-protein therapy. The picture was that of an increasingly severe conjunctivitis, ulcerative keratitis, and iritic involvement. The virus of lymphogranuloma was demonstrated by the inoculation of mice and positive Frei test. The patient was entirely well after eleven days of sulfadiazine therapy. (Bibliography, 3 figures.) George H. Stine.

Öylü, Lûtfi. **A case of ocular myiasis.** Türk Oft. Gazetesi, 1941, v. 3, pt. 10, p. 456. (French abstract, p. 474.)

Reports of two cases of larvae in the conjunctival sac. George A. Filmer.

Páez Allende, Francisco. **Ocular signs in two brothers with juvenile tabes and general paralysis.** La Semana Med., 1942, v. 49, March 12, pp. 491-499.

The patients were 22 and 24 years old respectively. One had marked anisocoria, mydriasis, pupillary deformity, complete lack of reaction to all light, and a hardly visible reaction of the right pupil to accommodation. The second brother had maximum mydriasis, slight anisocoria, complete abolition of light reaction, and almost complete lack of reaction to accommodation. Both brothers had good distant and near vision. The Wassermann reaction was negative in each case. (4 illustrations.) W. H. Crisp.

Pendse, G. S. **Allergy in ophthalmology.** Indian Jour. Ophth., 1941, v. 2, July, p. 90.

A brief description of the development of our concepts regarding allergy and immunity is given and the following ocular conditions are listed as being of allergic origin: allergic and neuroallergic conjunctivitis, phlyctenular keratoconjunctivitis, spring catarrh, episcleritis, endophthalmitis phakoanaphylactica, atopic cataract, allergic endophthalmitis, intraocular tuberculosis, and syphilis. Treatment is outlined as: (1) avoidance of antigen, or (2) desensitization to specific allergens.

Edna M. Reynolds.

Pimentel, P. C. **Consanguinity and morbid heredity.** Ophtalmos, 1941, v. 2, no. 2, pp. 329-334.

The author has had opportunity to study a number of members of a family which includes four cases of hydrophthalmos. The marriage of cousins is common in this family, and so also are ocular disorders. The family record is given in detail, with a genealogic tree. The four cases of hydrophthalmos are the products of two marriages of first cousins. The numerous marriages of more remote cousins yielded only cases of myopia. The total number of consanguineous marriages was 24. No other form of ocular anomaly was encountered. (References.)

W. H. Crisp.

Sottini, Giovanni. **Contribution to the study of palpebral cutaneous leishmaniasis.** Boll. d'Ocul., 1940, v. 18, March, pp. 223-242.

A woman of 25 years showed an irregularly oval neoformation in the skin near the external canthus. The growth was reddish-brown in color and of elastic consistency. It was movable on the underlying tissues and was surrounded by an edematous reddish zone. A crust in the central portion covered a depression filled with grayish exudate. This neoformation represented a relapse of a similar growth which had been removed a few months previously. Microscopic examination of the exudate showed the presence of leishmaniasis. (Bibliography, 2 figures.)

M. Lombardo.

Vazquez Barrière, and May, J. **Ocular manifestations in poradenolymphitis or lymphogranuloma venereum.** Arch. de Oft. de Buenos Aires, 1941, v. 16, Jan., p. 1.

The authors report their findings in the ocular examination of 120 patients affected with lymphogranuloma venereum. The full picture of the ocular syndrome revealed marked visibility and abnormal tortuosity of the finer nerve trunks of the central portion of the cornea, with increase in the opalescence and optical density of the stroma. The edges of the papilla were blurred and surrounded by a grayish edema of the neighboring retina. The veins were dilated and dark in color and their course was very tortuous. The intraocular tension was decreased and the retinal blood pressure showed definite hypertension. When the syndrome is incomplete some of these ophthalmoscopic signs may be absent, but papilledema and ocular hypotension are usually present in all cases. The ocular manifestations are found not only in the acute stages but also in the chronic phases of the disease. The eye examination may be negative in the early stages but ophthalmoscopic signs are usually evident about 15 days after the appearance of the chancre. The authors believe that the papilledema and venous stasis are related to increased intracranial pressure. In the light of their experience, the ocular manifestations, observed in 80 percent of the cases affected with lymphogranulomatosis venereum, are of considerable diagnostic value, particularly in the presence of ocular hypotension, which they found in 87.5 percent of the patients with ocular involvement. Plinio Montalván.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Bruce, G. M. **A note on penal blinding in the middle ages.** Annals of Med. History, 1941, v. 3, Sept., p. 369.

The author cites many instances in which blinding was used as a penalty for offense against the land.

Theodore M. Shapira.

Hamilton, J. B. **Eye diseases found in Tasmania: an eight-year survey.** Med. Jour. Australia, 1942, v. 1, Jan. 10, p. 45.

This article is a statistical résumé of the diagnoses in 6,460 eye cases in Tasmania for the years 1931 to 1939. The list includes 239 individual diseases. The author makes the following comments: Tuberculosis was probably the cause of a large percentage of the 127 cases of old choroiditis. There were only three cases of embolism of the central artery, compared with 31 cases of thrombosis of the central vein. The small figure for the former is, the author believes, due to the low incidence of rheumatic endocarditis in Tasmania. There were 29 birth injuries and 39 cases of ptosis. There were two cases of retinal glioma, four cases of coloboma of the choroid, one case each of aniridia and of coloboma of the iris. There were no cases of buphthalmos.

Ralph W. Danielson.

Kuhn, H. S. **An appraisal of visual defects in industry.** Trans. Amer. Acad. Ophth. and Otolaryng., 1941, 45th mtg., May-June, p. 208.

A total of 16,332 employees were tested as to visual acuity with and without the employee's correction, stereopsis ratings, color vision, and muscle balance for distance and near. The author emphasizes the importance of binocular testing of visual performance in industry. Many improvements and safeguards as to accuracy have been worked out. Complete examination of an employee's eyes should be made when he is hired. While surveys and visual-performance tests may be administered to better advantage by the trained lay expert, the entire program should be under the supervision of an ophthalmologist. Kuhn advises the sponsoring (by the Academy) of a program on industrial problems pertaining to vision, which could be worked out by a committee. (14 figures, discussion.)

George H. Stine.

Loewenstein, Arnold. **The education of an ophthalmic surgeon.** Brit. Jour. Ophth., 1942, v. 26, Feb., p. 45.

The author believes that a complete psychologic report should accompany every application to enter the study of medicine, such a report to cover speed of reaction, general intelligence, motor coördination and temperament. The following outline is recommended for preparation in ophthalmology: (1) study of histologic and bacteriologic method for three months; (2) three months of physiology with attention to theory and to experimental method; (3) six months devoted to general surgery to learn asepsis, use of various instruments, and suture technique. Large scholarships granted by government or by private charity are recommended to make this type of preparation possible for students not having independent means.

The author feels that the continental system of full-time assistantships is best for clinical education. He recommends creation of full-time assistantships on pay, and with private practice prohibited. The 26-hour undergraduate course in ophthalmology given in British medical schools is contrasted with the continental training period of 320 hours. The British courses are considered so short as to be dangerous. The importance of visiting various clinics is emphasized as essential to completion of the ophthalmic surgeon's education. Edna M. Reynolds.

Mayer, L. L. **Eyesight in industry.** Arch. of Ophth., 1942, v. 27, Feb., pp. 375-405.

The author discusses the factors of illumination, visual acuity, glare, fatigue, eye muscles, colors, and contrasts in the practical examination of the eyes of employees in industry. He includes a discussion of medical care of the eyes and protection against hazards, workmen's compensation laws, insurance carriers and self-insurers, free and part-pay ophthalmic clinics, and types of ocular service for industry. A summary of illumination standards and methods is also given.

J. Hewitt Judd.

Pritikin, R. I. **Ophthalmology under field conditions.** Illinois Med. Jour., 1941, v. 80, Nov., p. 373.

A timely report by an oculist in service.

Schoenberg, M. J. **Survey of records of glaucoma in ophthalmic clinics.** Arch. of Ophth., 1942, v. 27, April, pp. 716-727; also Trans. Amer. Ophth. Soc., 1941, v. 39, p. 161. (See Section 8, Glaucoma and ocular tension.)

Streiff, Giacomo. **Giovanni Battista Verle, Venetian: famous ivory turner in the service of Cosimo III grand duke of Tuscany, and anatomist of the human eye.** Ann. di Ottal., 1940, v. 68, Aug., pp. 629-638.

Verle, who in 1674 constructed a marvelously perfect anatomic model of the human eye in ivory, acquired European fame for the excellence of his work and design, which was according to nature rather than schematic, and won the praise of Morgagni. Quotations, with appropriate comments, are made from Verle's account of his dissection of the eye, and drawings showing the construction of his model are reproduced from a book published in 1680. Some historical notes on a celebrated doctor, Jacobus Wepfer, who was a contemporary of Verle, are included.

Harry K. Messenger.

Thau, William. **Purkyně: a pioneer in ophthalmoscopy.** Arch. of Ophth., 1942, v. 27, Feb., pp. 299-316.

The purpose of this report is to show that Purkyně was the first to announce the principle of ophthalmoscopy. Helmholtz is credited with putting Purkyně's idea to practical advantage by devising the eye mirror. Purkyně never exploited any of his discoveries, but was always searching for new knowledge and finding it. Thau is the first to submit the original text, together with the English translation, from Purkyně's thesis published in 1823, in which Purkyně describes almost exactly the procedure which thirty years later Helmholtz used for visualizing the interior of the eye.

J. Hewitt Judd.

Vila Ortiz. **Considerations concerning injuries of the cornea due to industrial accidents.** Arch. de Oft. de Buenos Aires, 1941, v. 16, Jan., p. 19; Feb., p. 70; March, p. 130; and April, p. 180. (See Section 16, Injuries.)

Wheeler, M. C. **The history of orthoptics.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 569-574.

PAN-AMERICAN NOTES

Edited by Dr. M. Uribe Troncoso
500 West End Avenue, New York

Communications should reach the Editor by the twelfth of the month

Miscellaneous

The Interamerican Session, heralded as one of the highlights of the annual meeting of the American Medical Association in June, was another manifestation of the increasingly close ties which link the Americas. The trend to better relations with Latin America is one of the few beneficial effects of the war to date. This tendency has been strong in medicine, particularly in New York. This Session provides additional evidence of the strength of the Good Neighbor Policy among the people of the United States. The physicians assembling in Atlantic City from all parts of the country welcomed this opportunity to get acquainted with some of their Latin-American colleagues and share their professional experiences and views.

Societies

A list of ophthalmological societies in Latin America with names and addresses of presidents is given:

Sociedad de Oftalmologia de Buenos Aires: Presidente Dr. Juan Gallino—740, Viamonte—Buenos Aires, Argentina.

Sociedad de Oftalmologia del Litoral: Presidente Prof. Carlos Weskamp—1159, Laprida—Rosario, Argentina.

Sociedad de Oftalmologia de Cordoba: Presidente Dr. Urretz Zavalia—255, 27 de Abril—Cordoba, Argentina.

Sociedade Brasileira de Ophthalmologia: Presidente Prof. O. Rego Lopes—99, Siete de Septiembre—Rio de Janeiro.

Sociedade de Ophthalmologia de São Paulo: Presidente Dr. W. Belfort Mattos—Barao de Itapetininga 297—São Paulo.

Sociedade de Ophthalmologia de Recife: Presidente Dr. Gilberto Fraga—49, Vis. Rio Branco—Recife, Estado de Pernambuco.

Sociedade Bahiana de Ophthalmologia: Presidente Dr. Theonilo Amorim—Barra Avenida, Bahia, Brazil.

Sociedade de Ophthalmologia de Bello Horizonte: Presidente Dr. Humberto Martins Vieira—Hospital dao Vicente—Bello Horizonte, Estado de Minas Geraes, Brazil.

Sociedade de Ophthalmologia de Porto Alegre: Presidente Dr. Santavana Mascarenhas—Galeria Chaves—Porto Alegre, Estado de Rio Grande do Sul, Brazil.

Sociedad Chilena de Oftalmologia: Presidente Prof. Carlos Charlin—660, Florestal—Santiago, Chile.

Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia: Presidente Dr. Puig Solanes—Palma 10—Mexico D. F.

Sociedad de Oftalmologia del Perú: Presidente Dr. Jorge Valdeavellano—149 Mineria—Lima, Perú.

Sociedad de Oftalmologia del Uruguay: Presidente Dr. Washington Isola—1218, Uruguay—Montevideo, Uruguay.

Sociedad de Otolaringologia, Oftalmologia y Neurologia: Secretario Dr. F. Conde Jahn—Apartado 1428—Caracas, Venezuela.

Personals

In his inaugural address, Dr. Maximilian A. Ramirez, president of the Medical Society of the County of New York, urged the development of closer relations with South American physicians by increasing the opportunities for graduate study in the metropolitan area, improving its organization, and lowering its cost. Shortly thereafter the New York Academy of Medicine announced the establishment of an Inter-American Division to cultivate better understanding among the medical practitioners of the nations of the Western hemisphere. The Bureau of Clinical Medicine took practical steps to implement this policy by placing its facilities at the disposition of visiting Central and South American physicians.

Journals

Anales de la Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia (address, Escuela Nacional de Medicine, esq. Brasil y Venezuela, Mexico, D. F.), 1941, volume 16, number 4. Eye articles: Elías Mendoza Gonzalez, Optic neuritis in pellagra; Abelardo Zertuche, Exenteration of the eyeball with implantation of fat; R. Mendiola, A case of cervical lymphogranulosis (disease of Nicolas and Favre). Also proceedings of the Mexican Society of Ophthalmology.

Archivos de Oftalmologia de Buenos Aires (address, Peru 795, Buenos Aires, Argentina), 1941, volume 16, number 11. Alberto Urrets Zavalia and Roberto Obregon Oliva, Case reports of Marcus Gunn phenomenon; Alberto Urrets Zavalia and Roberto Obregon Oliva, New considerations as to corneal chrysosis; Roberto Obregon Oliva, Agustin Caeiro, and Ignacio Ferreyra Videla, Radiographic lung

pictures in ocular tuberculosis; Jorge A. Peyret, Iontophoresis and catholysis in ophthalmology (continued).

Issue of 1941, volume 16, number 12. A Vázquez Barriere, The eye in Nicolas-Favre disease; Diego M. Arguello, Poerio Lambre, and Bruno Tosi, A case of Crouzon's disease; José A. Sena, Trachoma among the school children of Mendoza; Virgilio A. Victoria, A classification of the clinical forms of Argentinian trachoma; Jorge A. Peyret, Iontophoresis and catholysis in ophthalmology (continued). Each issue of the Archivos also contains a brief account of the proceedings of the Ophthalmological Society of Argentina.

Arquivos Brasileiros de Oftalmologia (address, Caixa Postal, 4086, São Paulo, Brasil), 1941, volume 4, number 6. Contents: Hilton Rocha, Atypical association in a macular coloboma with abnormal vessels; I. E. Barhel, A. A. Vichnevki, L. A. Denichitz, R. Kh. Mükaelian, and V. V. Tchirkovski, Nervous trophism of keratitis; Durval Prado, From Wollaston to Javal; Sergio Valle, Auto-operation for cataract; V. Hanke, Treatment of trachoma with albucid. Also proceedings of Brazilian ophthalmological societies.

Ophthalmologia Ibero Americana (address, calle Florida, 229 Buenos Aires, Republica Argentina U.T. 33 Avenida 1125), 1941, volume 3, number 3. Contents: Harry S. Gradle, Capsular glaucoma. Also 45 pages of abstracts from the world literature of ophthalmology.

Archivos Venezolanos de la Sociedad de Oto-Rino-Laringologia, Oftalmologia, Neurologia (Caracas, Venezuela), 1941, December. Eye articles: José Izquierdo, Crossing of the nerve paths and nerve mechanism of the ocular movements; Francisco Ramirez, Superficial keratectomy (bibliography).

NEWS ITEMS

Edited by Dr. Ralph H. Miller
803 Carew Tower, Cincinnati

News items should reach the editor by the twelfth of the month

Deaths

Dr. Philip Allen Halper, Chicago, Illinois, died April 21, 1942, aged 44 years.

Dr. M. Russell Wilcox, Los Angeles, California, died March 25, 1942, aged 73 years.

Dr. John Ernst Barnstein, Manitowoc, Wisconsin, died April 20, 1942, aged 89 years.

Dr. Walter E. Spicer, Jackson, Michigan, died March 8, 1942, aged 86 years.

Dr. Max R. Dinkelspiel, New York, New York, died March 13, 1942, aged 66 years.

Dr. John William Thomason, Huntsville, Texas, died March 4, 1942, aged 77 years.

Dr. William Joseph Riordan, Wallingford, Connecticut, died in March, 1942, aged 56 years.

Dr. Thomas Jefferson Halsey, Butler, Missouri, died February 26, 1942, aged 74 years.

Dr. Garrett M. VanAusdall, New London, Iowa, died February 27, 1942, aged 83 years.

Dr. Herman Burgess Cole, Redwood Falls, Minnesota, died March 29, 1942, aged 70 years.

Dr. Robert Dickson Ferguson, Pilot Mound, Manitoba, Canada, died March 2, 1942, aged 75 years.

Dr. William P. Wherry, Omaha, Nebraska, died June 14, 1942, aged 62 years.

Dr. John DeWitt Byrne, DuQuoin, Illinois, died April 12, 1942, aged 62 years.

Dr. Thomas Richard Luther, Lawton, Oklahoma, died March 7, 1942, aged 56 years.

Dr. Norven Henry Gillespie, Duluth, Minnesota, died March 26, 1942, aged 67 years.

Dr. Albert Joseph LaFrance, Laconia, New Hampshire, died March 12, 1943, aged 70 years.

Dr. Virgil C. Littlefield, Nixon, Texas, died recently, aged 70 years.

Dr. Samuel Arthur Looper, Hot Springs, Arkansas, died March 23, 1942, aged 67 years.

Miscellaneous

The Leslie Dana Gold Medal, awarded annually for outstanding achievements in the prevention of blindness and the conservation of vision, was presented this year to Lewis H. Carris, Director Emeritus of the National Society for the Prevention of Blindness. This highly prized token of recognition in the field of public health is given upon the recommendation of the Association for Research in Ophthalmology.

Societies

The New York Society for Clinical Ophthalmology elected the following officers for the year 1942-1943: Dr. Isadore Givner, president; Dr. Sigmund A. Agatston, vice-president; Dr. Jesse M. Levitt, recording secretary; Dr. Benjamin Esterman, corresponding secretary; and Dr. Daniel Kravitz, treasurer.

Among the guest speakers at the meeting of the Pittsburgh Ophthalmological Society on

April 27th were Dr. Louis W. Statti who spoke on "Pentothal sodium anesthesia in ophthalmology," and Dr. Edmund B. Spaeth who discussed "Treatment of chronic dacryocystitis."

At the seventy-eighth annual meeting of the American Ophthalmological Society held in Hot Spring, Virginia, June 1st, 2nd, and 3rd, the following officers were elected: Dr. Hunter H. McGuire, president; Dr. John Green, vice-president; and Dr. Wilfred E. Fry, editor of the Transactions. The council members include Dr. J. Milton Griscom, Dr. John H. Dunnington, Dr. Sanford R. Gifford, Dr. Parker Heath, and Dr. Lawrence T. Post. The members of the committee on theses are Dr. John A. McMillan, Dr. Phillips Thygeson, and Dr. John W. Burke. The program committee consists of Dr. Walter I. Lillie, Dr. Hunter H. McGuire, and Dr. Walter S. Atkinson.

The twenty-fourth annual meeting of the North Dakota Academy of Ophthalmology and Oto-laryngology was held on May 19th. The program consisted of an address by Dr. Lawrence R. Boies of the University of Minnesota, and the presentation of a case report, entitled "Cholesteatoma of the orbit," by Dr. George M. Constans. Following the program a business session was held. The following officers were elected for the coming year: Dr. G. A. Larson, president; Dr. T. W. Buckingham, vice-president; Dr. F. L. Wicks, secretary-treasurer; and Dr. M. B. Ruud, member of the Council.

Personals

Dr. Walter Guernsey Fry was among the guest speakers at the one-hundred-fiftieth anniversary meeting of the Connecticut State Medical Society. The title of his paper was "Elisha North, pioneer Connecticut ophthalmologist."

At the annual meeting of the Arizona State Medical Association Dr. William A. Boyce spoke on "Anesthesia and procedure of safety in eye operations."

The Pray and Burnham prizes are awarded annually to the authors of the two best essays submitted by members of the New Hampshire Medical Society during the past year. The prize is announced at the annual meeting of the New Hampshire Medical Society by its trustees. This year the first prize of $100.00 was given to Dr. Arthur Linksz of the Dartmouth Eye Institute, in Hanover, for an essay entitled "Dermatotherapy as applied to diseases of the eyelids," as announced at the annual meeting of the New Hampshire Medical Society in Manchester, on May 13th.

Dr. Eugene Chan has been recently appointed visiting professor of ophthalmology at the Central University of Medicine and has also been commissioned colonel in the Chinese Army Medical Corps. For the last three years he has held the chair in ophthalmology at West China University and has been head of the eye service of the Chengtu Eye, Ear, Nose, and Throat Hospital.